# HISTOPATHOLOGICAL STUDY OF INTRACRANIAL LESIONS

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I hereby declare that the thesis entitled "**HISTOPATHOLOGICAL STUDY OFINTRACRANIAL LESIONS**" submitted towards partial fulfillment of the requirements for the award of **MD in Pathology** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. HarpreetKaur**, Professor, Department of Pathology, **Dr. KavitaSaggar**, Professor&Head,Department of Radiodiagnosis and **Dr.VikramNarang**, Associate Professor, Department of Pathology. No part of this thesis has formed the basis for the award of any degree previously.

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*Dedicated*

*to*

*my Family*

*&*

*Teachers*

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**JANUARY, 2022** **DR. GAGANDEEP SINGH**

# ABSTRACT

**Introduction** - Intracranial space occupying lesions [ICSOL's] are a variety of conditions that can range from inflammatory to infectious to malignant. The histological spectrum of non-neoplastic and neoplastic lesions is broad, and it varies between extremes of age groups as well as between genders.

**Objectives**-

1. To study various histological patterns of Intracranial lesions.
2. To correlate histopathological diagnosis with radiological findings (wherever possible).

**Materials and methods**-The present study was a prospective and retrospective study on histopathological spectrum of intracranial lesions of specimens received in the Department of Pathology, Dayanand Medical College & Hospital, Ludhiana over a period of 5 years (3.5 years retrospectively from 1st July 2016 to 31st December 2019 and 1.5 years prospectively from 1st January 2020 to 30th June 2021). During this period all the specimens were analyzed with respect to light microscopic and IHC findings (wherever possible).

**Results**- A total of 71,244 specimens were received in the department, out of which 266 wereintracranial lesionsconstituting 0.37%. These specimens were classified after light microscopy and using immunohistochemistry wherever possible. Out of the 266 ICSOL's, 82.71% [220 cases] were neoplastic and 17.29% [46 cases] were non-neoplastic lesions with mean age of 48.5 years. Out of 220 neoplastic cases126 were males and 94 were females with M:F ratio of 1.3:1 and mean age of 49.2 years.Astrocytic tumors were the commonest neoplastic ICSOL's constituting 36.45% of total primary brain tumors, followed by meningioma 25.60% and pituitary adenoma 10.80%. Glioblastoma formed the major bulk with 60 cases. Frontal lobe being the overall commonest site for all ICSOL's. IHC was used in 42 cases with basic immunohistochemical markersi.e GFAP,IDH-1, ATRX, P53 and MIB-1. Other IHC markers were also used according to the need.

**Conclusion** –The current study examines the spectrum and frequency of various ICSOLs and concludes that when combined with IHC, radiological, and clinical findings, histological examination of biopsies is the gold standard for accurate diagnosis of various intracranial lesions.



**Keywords**- intracranial space occupying lesions, Brain tumors,Histopathology, Immunohistochemistry.

# LIST OF ABBREVIATIONS

| | |
|---|---|
| ICSOL | Intra Cranial Space Occupying Lesion |
| CNS | Central Nervous System |
| WHO | World Health Organization |
| IHC | Immunohistochemistry |
| IDH | Isocitrate Dehydrogenase |
| CSF | Cerebrospinal Fluid |
| CT | Computed Tomography |
| MRI | Magnetic Resonance Imaging |
| DWI | Diffusion-Weighted Imaging |
| OS | Overall Survival |
| PFS | Progression Free Survival |
| PML | Progressive Multifocal Leukoencephalopathy |
| AVM | Arteriovenous Malformation |
| HIV | Human Immunodeficiency Virus |
| GBM | Glioblastoma |
| GFAP | Glial Fibrillary Acidic Protein |
| ATRX | AlphaThalassemia Mental Retardation Syndrome-X |
| HPF | High Power Field |
| EMA | Epithelial Membrane Antigen |
| NSE | Neuron-Specific Enolase |
| MPNST | Malignant Peripheral Nerve Sheath Tumor |
| PCNSL | Primary Central Nervous System Lymphoma |
| CK | Cytokeratin |
| PPV | Positive Predictive Value |
| NPV | Negative Predictive Value |
| DPX | Distyrenedibutylphtalate xylene |

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# INTRODUCTION

Intracranial space occupying lesions [ICSOL] are the leading cause of neurological morbidity and mortality. ICSOL's can be neoplastic and non-neoplastic. Even when they are inflammatory lesions or benign neoplasms, they can have a serious clinical course.[1] The reason for this is that these lesions grow in a small space and are close to vital organs.

The location is also crucial because any lesion in Central Nervous System [CNS], regardless of histologic classification, can be fatal if it occurs in a critical brain region. Interpretation of histopathology of CNS tumors is significantly aided when viewed in perspective of clinical, per-operative and radiological findings.[2] Histopathology being the gold standard for diagnosing ICSOL's, radiological findings can help narrow down the possibilities. Because a brain biopsy is an invasive procedure, every effort should be made to arrive at a precise diagnosis prior to the procedure. [1]

Bacterial, fungal, viral, parasitic, ischemic, and inflammatory disorders are the different types of non-neoplastic lesions. Differentiating between brain tumors and tumor-like lesions is critical for determining appropriate treatment and estimating outcome and prognosis.[3] Neuroimaging plays a critical role in determining the correct distinction between the two entities. Intracranial tuberculosis is the common infective manifestation of central nervous system (CNS) and involvement in immunocompetent patients is the major cause of death and disability in developing countries due todiagnostic difficulties.[4] India together with Bangladesh, China, Pakistan and Indonesiaaccounts for half of the new cases arising each year.[5]

Intracranial hemorrhages can happen for a variety of factors, both intrinsic and extrinsic.[6] Extrinsic causes include accidental head trauma as well as surgical interventions. Intrinsic causes include hemorrhagic stroke, vascular malformations, aneurysms, hematological disorders, bleeding diathesis, anticoagulation treatment, intracranial neoplasms, dural sinus thrombosis. Transformation of hematomas may have

imaging features that look a lot like tumors. During the subacute to chronic phase, the spontaneous growth of a clot with contrast enhancement around it and the surrounding edema can mimic a tumor. The saccular or "berry" aneurysm is the most common type of cerebrovascular abnormality that can be surgically fixed. It is also the most common cause of massive subarachnoid hemorrhage in adults. These lesions are more common in women and happen at all ages after puberty, but they're more common in the fifth to seventh decades of life. Smoking, high blood pressure, aging, and having a positive family history are all risk factors. After a rupture, the death rate can be as high as 50%.[7,8]

Vascular malformations of the central nervous system (CNS) include a wide range of arterial and venous anomalies with varying presentations, clinical courses, and complication rates.[9] Arteriovenous shunts and cavernous malformations are the most common brain vascular lesions in adults, with detection rates of 1.0 and 0.5 per 100 000 adults per year, respectively.[10,11] Other vascular malformations are capillary telangiectasia and venous or cavernous angiomas.

A brain abscess is an Intrparenchymal collection of pus. Brain abscesses account for approximately 8% of intracranial masses in developing countries and 1–12% in western countries.[12,13] They start as localized areas of cerebritis in the parenchyma and progress to pus collections surrounded by a well-vascularized capsule. Bacterial infection from the paranasal sinuses is a common source of transmission.[14] The formation of an intracranial abscess is a direct result of the offending microorganism's virulence and the host's immune response. It is a serious, life-threatening disease and remains a fatal entity.[15,16] In later phases of contrast CT, uniform ring enhancement is almost always present. In the early stages, the capsule will be difficult to visualise using conventional techniques, and double contrast CT is frequently helpful in defining abscess encapsulation.[17] The use of 99m TC-hexamethylpropylene amine oxime leukocyte scintigraphy, diffusion weighted MR imaging, Thallium-201 single photon emission computed tomography, and

proton magnetic resonance spectroscopy (MRS) can help distinguish between abscess and tumor.[18-21]

Tumors of neuroepithelial tissue, peripheral nerves, meninges, mesenchymal, non-meningothelial tumors, lymphomas, germ cell tumors, and metastatic deposits are the World health organization's [WHO] histological classifications for nervous system neoplasms.[22] Tumors of the central nervous system are a rare occurrence, accounting for less than 2% of all malignancies. They primarily affect young adults, resulting in a higher global disease burden and, when compared to other tumors, a significant impact on disease adjusted life years.

Gliomas are the most common malignant primary brain tumors, accounting for 80% of all malignant primary brain tumors. Low-grade astrocytoma, oligodendroglioma, and mixed tumors, for example, have been found to progress to high-grade tumors over time. The length of time depends on the tumor's genetic and morphological makeup. Also Glioblastoma [GBM] is themost common primary brain tumor, with aggressive clinical manifestations.[23,24] Except for Turcot's syndrome andLi–Fraumeni syndrome, most GBM patients originate in a sporadic fashion without any known predisposing factors.[25] Compared with male patients, female patients with GBM have a higher cancer-specific survival after surgery.[24]

Meningiomas are the most common benign intracranial tumor in adults, accounting for nearly a third of all primary CNS tumors. Meningiomas have an annual incidence of 8.3 per 100,000 people and tend to rise with age.[25,26]

The WHO's 2016 classification of CNS tumors is largely a revision of its predecessor from 2008. The approach to CNS tumors, particularly gliomas, has changed dramatically in this edition. Isocitrate dehydrogenase [IDH] has been assigned to these gliomas as a molecular signature that aids in diagnosis and prognosis. Based on histopathological assessment and IDH mutation status, all diffusely infiltrating gliomas have been grouped together.[27] According to recent studies, having an IDH mutation is associated with a better prognosis,

with evidence of increased overall and progression-free survival. This is because the IDH mutation makes astrocytic tumors more chemotherapy and radiotherapy sensitive.

The presence of the IDH mutation distinguishes primary glioblastoma [GBM] from secondary glioblastoma, as it is predominantly positive in the latter. This further enhances IDH's prognostic value by detecting secondary glioblastomas, which are more common in younger patients than IDH-negative [wild type] primary glioblastomas.

Although the concept of immunohistochemistry [IHC] has been known since the 1930s, the first IHC study was published in 1942. The basic idea behind IHC is that specific antibodies are used to demonstrate antigen within tissue sections. Immunohistochemical markers, in addition to relevant clinical data, imaging techniques, and preoperative findings, have become necessary for a precise diagnosis and subtyping of brain tumors.

# AIMS AND OBJECTIVES

1. To study various histological patterns of Intracranial lesions.
2. To correlate histopathological diagnosis with radiological findings (wherever possible).

# REVIEW OF LITERATURE

The central nervous system [CNS], consists of the brain and spinal cord, is surrounded by meninges and suspended in a fluid called cerebrospinal fluid [CSF], which is produced by specialized choroid plexus structures.[28] All parts of the CNS are composed of grey and white matter on a macroscopic level. The majority of neuron cell bodies and dendritic processes are found in grey matter, while axons are found in white matter.

Any lesion, benign or malignant, primary or secondary, as well as any inflammatory or parasitic mass lying within the cranial cavity, is referred to as an intracranial space occupying lesion [ICSOL]. Hematomas, various types of cysts, and vascular malformations are also included.[29] Lesions that occupy intracranial space are a major cause of neurological morbidity. Their potentially lethal behavior stems from their confinement in a small space as well as their proximity to vital structures.The unparalleled complexity of the central nervous system (CNS) is reflected in the ever-increasing diversity of identifying neoplastic entities that can afflict the organ.[30,31]

Early diagnosis is required in order to plan effective intervention. To detect the lesion, radiological investigative modalities such as computed tomography [CT] and magnetic resonance imaging [MRI] are important preoperative diagnostic tools.[32] The goals of diagnostic imaging in patients with suspected intracranial masses include detecting the presence of a mass, localizing the extent of the mass [including defining the involvement of key structures and assessing the presence and severity of secondary changes such as edema, herniation, and hemorrhage, and characterizing the nature of the process.

**Normal brain embryology & histology**

The nervous system develops from neuroectoderm, a specialised ectoderm that overlies the notochord. Apart from blood vessels and some neuroglial elements, it is the source of the entire nervous system. The developing brain cavity has three dilatations craniocaudally: the

prosencephalon, mesencephalon, and rhombencephalon. The prosencephalon is divided into telencephalon and diencephalon, from which cerebral hemispheres, thalamus, hypothalamus, and epithalamus emerge. The mesencephalon helps to form the midbrain, while the rhombencephalon helps to form the pons, cerebellum, and medulla oblongata.[33]

The developing brain's subdivisions enclose a portion of the neural tube's original cavity. Each telencephalic vesicle's cavity becomes the lateral ventricle. The diencephalon cavity becomes the third ventricle, and the two lateral ventricles communicate with it via the interventricular foramen of monro. The cavity of the mesencephalon remains narrow and forms the aqueduct of Sylvius, which connects the third and fourth ventricles.[33] The fourth ventricle is formed by the cavity of the rhombencephalon. The spinal cord's central canal is the downward continuation of the fourth ventricle.

CSF is formed in the ventricles, primarily in the lateral ventricle, by choroid plexuses and flows from the lateral ventricles to the third ventricle via the interventricular foramen and from the third ventricle to the fourth ventricle via the cerebral aqueduct. It exits the ventricular system via a median foramen of magendie and two lateral foramen of luschka into the subarachnoid space surrounding the brain and spinal cord.

The choroid plexus is a vascular structure that arises from the wall of each of the brain's four ventricles and is responsible for the production of CSF. It is continuously produced and reabsorbed from the subarachnoid space into the superior sagittal venous sinus via finger-like projections known as arachnoid villi. The choroid plexus capillaries and vessels are large, thin-walled, and sometimes fenestrated, lying in a fibrous core. The basal lamina supports the epithelial cells. A blood-CSF barrier is aided by continuous tight junctions [zonula occludens] between epithelial cells. CNS is anatomically divided into cerebral cortex, cerebellum, pons, medulla, and spinal cord. The brain and spinal cord are surrounded by three layers of supporting tissue known

as the meninges, which include the dura mater, arachnoid mater, and pia mater.

All parts of the CNS are composed of grey and white matter on a macroscopic level. The majority of neuron cell bodies and dendritic processes are found in grey matter, while axons are found in white matter. These are supported by a group of cells known as neuroglia, which includes astrocytes, oligodendrocytes, microglia, and ependymal cells.[28]

• Astrocytes are branched cells that exist between neurons and provide mechanical support as well as mediate metabolite exchange between neurons and the vascular system. Astrocytes are identified using immune-histochemical staining for glial fibrillary acidic protein [GFAP]

• Oligodendrocytes are the equivalent of Schwann cells and are in charge of myelination.

• Microglia are CNS monocyte-macrophage system representatives with defence and immunological functions.

• Ependymal cells are the building blocks of the specialised epithelium that lines the ventricles and spinal canal.

The CNS lacks collagenous supporting tissue, which is limited to the immediate surroundings of penetrating blood vessels and the meninges that invest the brain's outer surface.

White matter is made up of nerve fibers [axons] that have been myelinated by oligodendrocytes. They form tracts and are supported by astrocytes, microglia, and blood vessels. One oligodendrocyte can myelinate approximately 50 axons. Myelin sheath formation follows a similar mechanism to that of Schwann cells in peripheral nerve.

The cerebral hemispheres are made up of outer grey and inner white matter. Neurons in the cortex are classified into five types based on histology.

• Pyramidal cells – These are pyramid-shaped cell bodies with the apex pointing towards the cortical surface. The axon emerges from the cell's base and travels into the white matter. Betz cells, the largest pyramidal cells in the cortex, are the largest motor neurons of the motor cortex.[28]

• Stellate cells – Stellate cells are small neurons with a star-shaped body due to branching dendrites and a short axon. Found in the cortex's superficial layers.[28]

• Cells of Martinotti – These cells can also be found in the superficial layers. They are small polygonal cells with a few short dendrites and an axon that extends towards the surface before bifurcating to run horizontally.

• Fusiform cells – These are spindly-shaped cells that are oriented at 90 degrees to the cerebral surface. The axon emerges from the cell body's side and travels superficially.

• Horizontal cells of Cajal – These are small, spindly cells that are oriented parallel to the surface. They are the least common type of cell and can only be found in the most superficial layer.

The neocortex is divided into six layers, each with its own neuron morphology, size, and population density. It has the following layers:

1. Plexiform layer, 2. Outer granular layer, 3. Pyramidal cell layer, 4. Inner granular layer, 5. Ganglionic layer, and 6. Multiform cell layer.

The cerebellar cortex is made up of three layers: the molecular layer, the purkinje cell layer, and the granular layer. The molecular layer has a small number of neurons and a large number of unmyelinated fibers.[28] The deep granular cell layer of the cortex contains numerous small neurons, the non-myelinated axons of which pass outwards to the molecular layer, where they bifurcate to run parallel to the surface and synapse with purkinje cell dendrites.

The pons is the brainstem's middle portion, located between the midbrain and the medulla. It is divided into two sections: a large ventral region [basal pons] and a smaller dorsal [tegmental] region. The basal pons is made up of crisscrossed bundles of longitudinal and transverse fibers that connect to pontine nuclei, which are collections of neuron cell bodies.[28]

The substantia nigra is a large mass of grey matter that extends throughout the midbrain, dividing the cerebral peduncles into dorsal and ventral parts on each side. Neurons with dark pigment are easily identified histologically. The substantia nigra has extensive connections

with the cortex, spinal cord, corpus striatum, and reticular formation, and appears to play an important role in motor function fine control.

The brainstem's most caudal part, the medulla oblongata, connects to the spinal cord and contains numerous other ascending and descending tracts, as well as various tracts and nuclei of the eighth to twelfth cranial nerves. A spinal nucleus, which is part of the trigeminal nerve and its associated tract, is also found in the medulla. Cell bodies of lower motor neurons running in the spinal accessory and first cervical nerves are found in the ventral grey matter horns.

The brain and spinal cord are surrounded by three layers of supporting tissue known as the meninges. The nervous tissue is protected by the pia mater, a delicate layer composed of collagen fibers, fine elastin fibers, and occasional fibroblasts that is separated from the astrocytic processes of the underlying CNS parenchyma by a basement membrane.

Overlying the pia mater is a thicker fibrous layer known as the arachnoid mater, which gets its name from the presence of cobweb-like strands that connect it to the pia mater. The pia-arachnoid is also known as the leptomeninges. The space between the pia and the arachnoid is known as the subarachnoid space, and it can form large cisterns in some places.

The dura mater is a dense fibroelastic layer that surrounds the arachnoid mater and is lined on its internal surface by flat cells. The dura is close to but not connected to the arachnoid layer, and there is the possibility of a space, the subdural space. Denticulate ligaments suspend the dura from the periosteum of the spinal canal, with the intervening epidural space filled with loose fibrofatty tissue and a venous plexus.

**Epidemiology of intracranial lesions**

The ICSOL’s are subdivided into non-neoplastic and neoplastic lesions. Infarcts, bacterial or viral infections, intracranial cysts, brain abscesses, CNS mycoses, tuberculoma, amyloidosis, vasculitis, and hematomas

are examples of non-neoplastic lesions. Non-neoplastic lesions are far less common than neoplastic lesions. A wide range of non-neoplastic conditions, both clinically and radiologically, can mimic a brain tumor, posing a risk to physicians involved in patient care.[34] Differentiating between tumors and tumor-like lesions of the central nervous system [CNS] is critical for treatment planning as well as estimating outcome and future prognosis.

An intracranial abscess is the most common tumor-like lesion. Abscesses can develop as a result of a meningoencephalitis, as a direct extension of paranasal sinus and temporal bone infections, or as a result of hematogenous spread.[3] On conventional imaging abscesses are round, mass lesions with various degrees of adjacent white matter edema and a strong peripheral contrast enhancement. The identification of restricted diffusion within the center of the abscess facilitated differentiation from necrotic high-grade malignancies significantly with the advent of diffusion-weighted imaging [DWI].

In the United States, the annual incidence of CNS tumors ranges from 10 to 17 per 100,000 people for intracranial tumors and 1 to 2 per 100,000 people for intraspinal tumors.[35] CNS tumors are more common in children than in adults, second only to leukaemia. In India, approximately 87 % of CNS neoplasms occur intracranially, with spinal tumors accounting for the remaining 13 % of cases.

More than half of CNS tumors are primary tumors, with the remainder being metastatic.[35] Males have a slightly higher prevalence of primary brain tumors. They have a bimodal distribution, with a small peak in the pediatric population and a steady increase in incidence with age, beginning at the age of 20 years and peaking between the ages of 75 and 84.[36] East Asia had the highest number of new cases of CNS cancer for both sexes in 2016, followed by Western Europe and then South Asia. China, the United States, and India have the highest number of new cases of CNS tumors.[36]

Meningioma is the most common primary brain tumor in adults, accounting for roughly 37% of all primary brain tumors, followed by gliomas, pituitary tumors, nerve sheath tumors such as acoustic

neuromas, lymphomas, and embryonal tumors such as medulloblastoma.[37]

**Clinical presentation of CNS lesions**

- Clinical manifestation of an abscess: The clinical features depend on the host's immune status, specific pathogen involved, contiguous sites of infection or distant foci of infection, size of the abscess and location of the lesions. The most common symptom is headache, seen in about 70% of cases. Seizures are also a common first symptom, occurring in about 25-34% of patients.[14,38,39] Other common manifestations involve changes in level of consciousness, nausea, vomiting, high fever and neck stiffness.[14] A suddenly worsening headache followed by emerging signs of irritation of meninges are often associated with rupture of the abscess. An occipital abscess may rupture into ventricle causing ventriculitis or ependymitis or it may cause septic thrombophlebitis of the transverse sinus causing venous hypertension, edema, seizures and raised intra-cranial pressure.
- Clinical manifestations of the CNS tumors: The clinical features depend on the location and the growth rate of the tumors. Rapid enlargement of the tumor may cause rise in the pressure of the affected compartment and rise in intracranial pressure.[35]

  The signs and symptoms can be general, non-localizing and focal. Generalized symptoms include headache and seizures. Other symptoms include loss of consciousness, personality changes, amnesia, nausea, vomiting and fatigue. Symptoms that may be specific to the location of the tumor include.[35]

  - ❖ The cerebellar lesions present with clumsiness, nausea, vomiting, loss of balance and difficulty with fine motor skills.
  - ❖ Lesions in the hypothalamic area, thalamic area and internal capsule present with obstructive hydrocephalus in addition to obesity and sensory or motor deficit.

- Frontal lobe lesions cause changes in judgement including loss of initiative, sluggishness and muscle weakness or paralysis.
- Lesions in temporal lobe cause changes in speech, hearing, memory, or emotional state such as aggressiveness and problem of understanding or retrieving words.
- Parietal lobe lesions cause altered perception of touch or pressure, arm or leg weakness on one side of the body, or confusion with left and right sides of the body.
- Lesions in the optic pathway or occipital lobe present with visual loss or proptosis.
- Pituitary tumors cause lactation and menstrual irregularities in women.
- Tumor in the brain stem case difficulty in swallowing, facial weakness or numbness and double vision.
- Spinal cord involvement manifests with back pain, weakness or numbness in arms or legs and loss of bowel or bladder function

**Classification and grading of CNS tumors**

Early attempts to establish an internationally accepted, systematic approach to brain tumor nomenclature, such as the International Union Against Cancer, Atlas of the Histology of Brain Tumors and the Atlas of Gross Neurosurgical Pathology were unsuccessful. In 1952, James W. Kernohan and George P. Sayre's Armed Forces Institute of Pathology Fascicle for Tumors of the Central Nervous System had little success.[40]

Five different classifications of central nervous system tumors have been published. Zülch edited the first edition on the histological typing of tumors of central nervous system, which was published in 1979. Kleihues et al. edited the second edition in 1993. Immunohistochemistry was introduced into diagnostic pathology in this edition. Kleihues and Cavenee edited the third edition, which was published in 2000, and included genetic profiles of brain tumors. [40]

The 2007 edition includes a grading system that is parallel for each type of tumor. The biological behavior of a neoplasm is predicted by its histological grade. Grade I lesions have a low proliferative potential and

the possibility of cure after surgical resection alone. Grade II neoplasms are generally infiltrative in nature, and despite low levels of proliferative activity, they frequently reoccur. WHO grade III lesions have histological evidence of malignancy, including nuclear atypia and rapid mitotic activity. Tumors of WHO grade IV are mitotically active, have extensive necrosis, and are typically associated with rapid pre- and postoperative disease evolution and a fatal outcome.[27]

**Salient features of the 2016 WHO Classification of Tumors of the Central Nervous System:**

Many tumor entities are defined by molecular parameters in addition to histology in the 2016 WHO classification of CNS tumors. This classification of CNS tumors includes a significant restructuring of diffuse gliomas, medulloblastomas, and other embryonal tumors, as well as new entities defined by histology and molecular features.

The century-old principle of diagnosing tumors solely through microscopy has been broken, and the incorporation of phenotypic and genotypic parameters into the classification has added objectivity that has been lacking in certain diagnostics. This has resulted in more precisely defined entities, increasing diagnostic accuracy.

According to the new WHO classification of CNS tumors 2016, the presence of certain mutations like IDH in astrocytic tumors, co deletion of 1p-19q in oligodendroglial tumors are considered the major deciding factors of grading and prognosis. According to recent research, the presence of an IDH mutation is associated with a better prognosis, with evidence of increased overall and progression-free survival. This is because the IDH mutation increases the chemotherapeutic and radiotherapy sensitivity of gliomas.

In a meta-analysis published in 2016, Chen et al found that the presence of IDH1/2 mutations was associated with longer overall survival [OS] and progression free survival [PFS], and this result was seen in both surgical and radiotherapy patients. IDH1 mutations have been reported in secondary glioblastoma, diffuse astrocytoma, oligodendroglioma, anaplastic astrocytoma, anaplastic

oligodendroglioma, and anaplastic oligoastrocytoma, but not in pilocytic astrocytoma, ependymoma, or medulloblastoma.[41]

Nonneoplastic lesions of CNS

Infective Lesions

1. Bacterial infections
- Streptococcus
- Staphylococcus aureus
- Listeria
- Mycobacterium tuberculosis
- Escherichia coli
- Klebsiella- Enterobacter
- Hemophilus

2. Mycoses
- Aspergillus
- Candida
- Cryptococcus
- Coccidiodes
- Histoplasma
- Blastomyces

3. Parasitoses
- Taeniasolium
- Toxoplasma gondii
- Entamoeba histolytica

4. Spirochetal infections
- Treponema pallidum
- Borreliaburgdorferi

5. Viral infections
- Herpes simplex encephalitis
- Progressive multifocal leuco-encephalopathy
- Varicella zoster virus encephalit is and cerebral vasculitis
- HIV-1 encephalomyelitis

6. Prion associated diseases

Inflammatory lesions

- Demyelinating diseases
- Multiple sclerosis and its variants
- Noninfectious inflammatory and reactive disorders
- Xanthomatous lesions

Ischemic diseases

- Infarcts

**WHO CLASSIFICATION OF TUMORS OF THE CENTRAL NERVOUS SYSTEM 2016**

Diffuse astrocytic and oligodendroglial tumors
Diffuse astrocytoma, IDH-mutant
Gemistocytic astrocytoma, IDH-mutant
Diffuse astrocytoma, IDH-wildtype
Diffuse astrocytoma, NOS

Anaplastic astrocytoma, IDH-mutant
Anaplastic astrocytoma, IDH-wildtype
Anaplastic astrocytoma, NOS

Glioblastoma, IDH-wildtype
    Giant cell glioblastoma
Gliosarcoma
    Epithelioid glioblastoma
Glioblastoma, IDH-mutant
Glioblastoma, NOS
Diffuse midline glioma, H3 K27M-mutant

Oligodendroglioma, IDH-mutant and 1p/19q-codeleted
Oligodendroglioma, NOS

Anaplastic oligodendroglioma, IDH-mutant and 1p/19q-codeleted
Anaplastic oligodendroglioma, NOS
Oligoastrocytoma, NOS
Anaplastic oligoastrocytoma, NOS

Other astrocytic tumors
Pilocytic astrocytoma
Pilomyxoid astrocytoma
Subependymal giant cell astrocytoma
Pleomorphic xanthoastrocytoma
Anaplastic pleomorphic xanthoastrocytoma

Ependymal tumors
Subependymoma
Myxopapillaryependymoma
Ependymoma
  Papillary ependymoma
  Clear cell ependymoma
Tanycyticependymoma
Ependymoma, RELA fusion-positive
Anaplastic Ependymoma

Other gliomas
Chordoid glioma of the third ventricle
Angiocentric glioma
Astroblastoma
Choroid plexus tumors Choroid plexus papilloma
Atypical choroid plexus papilloma
Choroid plexus carcinoma

Choroid plexus tumors
Choroid plexus papilloma
Atypical choroid plexus papilloma
Choroid plexus carcinoma

Tumors of pineal gland
Pineocytoma
Pineal parenchymal tumor of intermediate differentiation
Pineoblastoma
Papillary tumor of the pineal gland

Embryonal tumors
Medulloblastomas, genetically defined
Medulloblastoma, WNT-activated
Medulloblastoma, SHH-activated and TP53-mutant
Medulloblastoma, SHH-activated and TP53- wildtype
Medulloblastoma, non-WNT/non-SHH
Medulloblastoma, group 3
Medulloblastoma, group 4
Medulloblastomas, histologically defined
Medulloblastoma, classic
Medulloblastoma, desmoplastic/nodular
Medulloblastoma with extensive nodularity
Medulloblastoma, large cell / anaplastic
Medulloblastoma, NOS
Embryonal tumor with multilayered rosettes, C19MC-altered
Embryonal tumor with multilayered rosettes, NOS
Medulloepithelioma
CNS neuroblastoma
CNS ganglioneuroblastoma
CNS embryonal tumor, NOS
Atypical teratoid/rhabdoidtumor
CNS embryonal tumor with rhabdoid features

Tumors of the cranial and paraspinal nerves Schwannoma
Cellular schwannoma
Plexiform schwannoma
Melanotic schwannoma
Neurofibroma
Atypical neurofibroma
Plexiform neurofibroma
Perineurioma
Hybrid nerve sheath tumors
Malignant peripheral nerve sheath tumor
Epithelioid MPNST
MPNST with perineurial differentiation

Meningiomas
Meningioma
Meningothelial meningioma
Fibrous meningioma
Transitional meningioma
Psammomatous meningioma
Angiomatous meningioma
Microcystic meningioma
Secretory meningioma
Lymphoplasmacyte-rich meningioma
Metaplastic meningioma
Chordoid meningioma
Clear cell meningioma
Atypical meningioma
Papillary meningioma
Rhabdoid meningioma
Anaplastic [malignant] meningioma

Mesenchymal, non-meningothelial tumors Solitary fibrous tumor / haemangiopericytoma
Grade 1, Grade 2, Grade 3
Haemangioblastoma
Haemangioma
Epithelioid haemangioendothelioma
Angiosarcoma
Kaposi sarcoma

Ewing sarcoma / PNET Lipoma
AngiolipomaHibernoma
Liposarcoma
Desmoid-type fibromatosis
Myofibroblastoma
Inflammatory myofibroblastictumor
Benign fibrous histiocytoma
Fibrosarcoma
Undifferentiated pleomorphic sarcoma / malignant fibrous histiocytoma
Leiomyoma
LeiomyosarcomaRhabdomyoma
Rhabdomyosarcoma
Chondroma
Chondrosarcoma
Osteoma
Osteochondroma
Osteosarcoma

Melanocytic tumors
Meningeal melanocytosis
Meningeal melanocytoma
Meningeal melanoma
Meningeal melanomatosis

Lymphomas
Diffuse large B-cell lymphoma of the CNS
Immunodeficiency-associated CNS lymphomas
AIDS-related diffuse large B-cell lymphoma
EBV-positive diffuse large B-cell lymphoma, NOS
Lymphomatoid granulomatosis
Intravascular large B-cell lymphoma
Low-grade B-cell lymphomas of the CNS T-cell and NK/T-cell lymphomas of the CNS Anaplastic large cell lymphoma, ALK-positive
lymphoma of the dura

Histiocytic tumors
Langerhans cell histiocytosis
Erdheim-Chester disease
Rosai-Dorfman disease
Juvenile xanthogranuloma
Histiocytic sarcoma

Germ cell tumors
Germinoma
Embryonal carcinoma
Yolk sac tumor
Choriocarcinoma
Teratoma
Mature teratoma
Immature teratoma
Teratoma with malignant transformation
Mixed germ cell tumor

Tumors of the sellar region
Craniopharyngioma
Adamantinomatouscraniopharyngioma
Papillary craniopharyngioma
Granular cell tumor of the sellar region
Pituicytoma
Spindle cell oncocytoma

Metastatic tumors

**WHO GRADES OF SELECT CNS TUMORS**

| | |
|---|---|
| Diffuse astrocytic and oligodendroglial tumors | |
| Diffuse astrocytoma, IDH-mutant | II |
| Anaplastic astrocytoma, IDH-mutant | III |
| Glioblastoma, IDH-wildtype | IV |
| Glioblastoma, IDH-mutant | IV |
| Diffuse midline glioma, H3 K27M-mutant | IV |
| Oligodendroglioma, IDH-mutant and 1p/19q-codeleted | II |
| Anaplastic oligodendroglioma, IDH-mutant and 1p/19q-codeleted | III |
| | |
| Other astrocytic tumors | |
| Pilocytic astrocytoma | I |
| Subependymal giant cell astrocytoma | I |
| Pleomorphic xanthoastrocytoma | II |
| Anaplastic pleomorphic xanthoastrocytoma | III |
| | |
| Ependymal tumors | |
| Subependymoma | I |
| Myxopapillaryependymoma | I |
| Ependymoma | II |
| Ependymoma, RELA fusion-positive | II OR III |
| Anaplastic ependymoma | III |
| | |
| Other gliomas | |
| Angiocentric glioma | I |
| Chordoid glioma of third ventricle | II |
| | |
| Choroid plexus tumors | |
| Choroid plexus papilloma | I |
| Atypical choroid plexus papilloma | II |
| Choroid plexus carcinoma | III |

| | |
|---|---|
| Neuronal and mixed neuronal-glial tumors | |
| Dysembryoplasticneuroepithelialtumor | I |
| Gangliocytoma | I |
| Ganglioglioma | I |
| Anaplastic ganglioglioma | III |
| Dysplastic gangliocytoma of cerebellum [Lhermitte-Duclos] | I |
| Desmoplastic infantile astrocytoma and ganglioglioma | I |
| Papillary glioneuronaltumor | I |
| Rosette-forming glioneuronaltumor | I |
| Central neurocytoma | II |
| Extraventricularneurocytoma | II |
| Cerebellar liponeurocytoma | II |
| | |
| Tumors of the pineal region | |
| Pineocytoma | I |
| Pineal parenchymal tumor of intermediate differentiation | II OR III |
| Pineoblastoma | IV |
| Papillary tumor of the pineal region | II OR III |
| | |
| Embryonal tumors | |
| Medulloblastoma [all subtypes] | IV |
| Embryonal tumor with multilayered rosettes, C19MCaltered | IV |
| Medulloepithelioma | IV |
| CNS embryonal tumor, NOS | IV |
| Atypical teratoid/rhabdoidtumor | IV |
| CNS embryonal tumor with rhabdoid features | IV |
| | |
| Tumors of the cranial and paraspinal nerves | |
| Schwannoma | I |
| Neurofibroma | I |
| Perineurioma | I |
| Malignant peripheral nerve sheath tumor [MPNST] | I, III OR IV |

| | |
|---|---|
| Meningiomas | |
| Meningioma | I |
| Atypical meningioma | II |
| Anaplastic [malignant] meningioma | III |
| | |
| Mesenchymal, non-meningothelial tumors | |
| Solitary fibrous tumor / haemangiopericytoma | I, II OR III |
| Haemangioblastoma | I |
| | |
| Tumors of the sellar region | |
| Craniopharyngioma | I |
| Granular cell tumor | I |
| Pituicytoma | I |
| Spindle cell oncocytoma | I |

**Morphology of common intracranial lesions**

- **NON-NEOPLASTIC**

**Cerebral infarction** - A sudden loss of neurologic function ["stroke"] caused by a narrowed arterial territory in the brain. The histology of a given infarct will depend on the stage of development.[42,43] Biopsied tissues typically have a spongy rarefaction that reflects the edema that is largely to blame for the associated mass effects of these lesions, as well as evidence of intense vascular congestion and may be openly haemorrhagic.[44]

**Aneurysm** - The rupture of a saccular "berry" aneurysm in a cerebral artery is the most common cause of spontaneous subarachnoid hemorrhage. Aneurysm's etiology is unknown, however the structural abnormality of the involved vessel suggests that they are developmental anomalies.

**Vascular malformations** - Arteriovenous malformations [AVM], cavernous malformations, capillary telangiectasias, and venous

angiomas are the four types of vascular malformations. Ectatic, variably muscularized blood vessels with interrupted elastic lamina and fibrotic intimal thickening characterize AVM.[44,45] Cavernous malformations are made up of distended, loosely organized vascular channels that are arranged back to back with variable thickness collagenized walls. There is usually no brain parenchyma between the vessels.

**Demyelinating diseases**– These are acquired conditions characterized by preferential myelin damage with relative axonal preservation. Multiple sclerosis causes immune-mediated myelin destruction, and infections cause progressive multifocal leukoencephalopathy [PML]. Affected tissues in multiple sclerosis exhibit diffuse infiltration by foamy, lipid-laden macrophages, reactive astrocytosis of variable intensity, and perivascular aggregates of small B and T lymphocytes.

Meningitis

**Acute pyogenic [Bacterial] meningitis**- On microscopic examination, neutrophils fill the subarachnoid space in severely affected areas and are primarily found around the leptomeningeal blood vessels in less severe cases. Gram stain reveals a variety of bacteria.

Acute Aseptic [Viral] Meningitis - Aseptic meningitis is a clinical term that refers to the absence of organisms by bacterial culture in a patient with manifestations of meningitis, such as meningeal irritation, fever, and altered consciousness of relatively recent onset.

**Brain abscess** – A brain abscess is a localized focus of necrosis with accompanying inflammation that is usually caused by a bacterial infection. Abscesses are distinct lesions characterized by a central liquefactive necrosis surrounded by brain swelling. There is exuberant granulation tissue with neovascularization at the necrotic lesion's outer margin.

**Cns mycoses** – It occurs most commonly in immunocompromised patients. Many are only discovered at autopsies. Fungal meningitis is commonly caused by Cryptococcus neoformans, Candida albicans, Coccidioidesimmitis, and Histoplasma capsulatum. Aspergillus species,

Candida species, Zygomycetes and some of the fungi are known to cause mass lesions in brain.

**Parasites** - Neurocysticercosis is the most common parasitic cerebral infection encountered worldwide, caused by larvae of the pork tapeworm, Taeniasolium. When present in the cerebral cortex, it most commonly causes focal or generalised seizures, but it can also cause obstructive hydrocephalus and elevated intracranial pressure when it colonises the ventricular system and basal cisterns. The fibrous pseudocapsule is typically infiltrated by numerous lymphocytes and plasma cells, may contain eosinophils in abundance, and in some cases is the site of an active granulomatous reaction, along with mummified remains of the degenerate scolex, which are covered by a wavy, refractile cuticle and consist largely of loose, reticular tissue containing numerous calcospherites.

**Cerebral toxoplasmosis** - It is an opportunistic infection commonly found in the setting of HIV-associated immunosuppression. Multiple ring-enhancing lesions may be seen on computed tomography and magnetic resonance imaging studies however, this radiographic appearance is not specific, as many other CNS lesions produce similar findings. Intracellular pseudocysts and "true" cysts filled with minute, PAS-positive bradyzoites are visible in biopsies, though in smaller numbers.

- **NEOPLASTIC**

**Astrocytoma** – A tumor that is moderately cellular and composed of well-differentiated fibrillary astrocytes. Nuclear atypia is a distinguishing feature, as is the absence of mitotic activity as well as necrosis. They correspond to WHO grade II histologically.[27]

Astrocytomas consistently express GFAP, and strong nuclear expression of p53 is also common. In contrast, alpha thalassemia mental retardation syndrome-X [ATRX] expression is almost invariably lost in the setting of ATRX mutations, which also features prominently in astrocytomas. They have a Ki-67 proliferation index of <4%.

Diffuse hypercellularity, rapid mitosis, giant cells, and a Ki-67 proliferation index of 5-10% are all indicators of anaplastic astrocytoma.

**Glioblastoma** – This is a high-grade glioma with predominantly astrocytic differentiation, nuclear atypia, cellular pleomorphism, mitotic activity, and typically a diffuse growth pattern, prominent microvascular proliferation, and/or necrosis.[27] GFAP is frequently expressed in glioblastomas, but the degree of reactivity varies greatly between cases for example, gemistocytic areas are always strongly positive, whereas primitive cellular components are frequently negative. S100, Nestin, and P53 are also frequently expressed. The Ki67 index is also significantly elevated.

The presence of the above features, as well as numerous multinucleated giant cells, small fusiform syncytial cells, and a reticulin network, leads to the diagnosis of giant cell glioblastoma.

**Gliosarcoma** – This type of glioblastoma has a biphasic tissue pattern with alternating areas of glial and mesenchymal differentiation. The separation of the two components is aided by the use of combined histochemical and immunohistochemical staining. The reticulin-free glial component is GFAP positive, while the mesenchymal component is negative. In almost all cases, IDH1 staining is negative. If p53 immunopositivity exists, it is found in both glial and mesenchymal components.

**Oligodendroglioma** - On routinely processed paraffin sections, this tumor is composed of cells that resemble oligodendrocytes morphologically, with isomorphic rounded nuclei and an artefactually swollen clear cytoplasm. Micro-calcifications are common, as is a delicate branching capillary network.

To date, no single immunohistochemical marker specific for oligodendroglial tumor cells has been identified. The majority of oligodendrogliomas show strong immunopositivity for IDH1, but a lack of IDH1 immunopositivity does not rule out the diagnosis. ATRX nuclear expression is typically retained in IDH-mutant and 1p/19q-

codeleted oligodendrogliomas. They are usually devoid of widespread nuclear p53 staining.

Anaplasia is associated with diffuse infiltration with prominent mitotic activity at least 6/10 high power field [HPF], microvascular proliferation, and focal necrosis.

**Oligoastrocytoma** - A diffusely infiltrating, slow-growing glioma composed of a prominent mixture of two distinct neoplastic cell types morphologically similar to tumor cells with either oligodendroglial or astrocytic features.

**Pilocytic astrocytoma** - It's a low to moderately cellular biphasic tumor with varying proportions of compacted bipolar cells with Rosenthal fibers and loose-textured multipolar cells with microcysts. Rare mitoses, hyperchromatic and pleomorphic nuclei, glomeruloid vascular proliferation, infarct-like necrosis, and leptomeningial infiltration are compatible with the diagnosis. They strongly express GFAP, S100, and OLIG2. In contrast to astrocytomas, p53 protein staining is weak to non-existent, which is consistent with the rarity of TP53 mutations. In most cases, the Ki-67 proliferation index is low.

**Pleomorphic xanthoastrocytoma** – The term "pleomorphic" refers to the tumor's variable histological appearance, in which spindled cells coexist with mononucleated or multinucleated giant astrocytes, and "xanthoastrocytoma" refers to the presence of large, often multinucleated xanthomatous cells with intracellular lipid accumulation. Also visible are reticulin fibers, which are best seen with silver impregnation method.

These tumors express GFAP and S100 due to their essential nature. They also have a high predilection for neuronal differentiation. Neuronal markers [such as synaptophysin, neurofilament, class III beta-tubulin, and MAP2][27] have been found to be expressed at varying levels. The BRAF V600E mutation is common and can be identified using immunohistochemistry. Ki-67 proliferation index is typically 1%.

Anaplasia in pleomorphic xanthoastrocytoma is typically characterized by brisk mitotic activity greater than 5/10 HPF, frequent necrosis, a high Ki-67 proliferation index, and no microvascular proliferation.

**Subependymoma** - It's a slow-growing, exophytic, intraventricular glial neoplasm characterized by clusters of bland to mildly pleomorphic, mitotically inactive cells embedded in a fibrillary matrix with frequent microcystic change. The fourth ventricle is the most commonly used location. GFAP immunoreactivity is usually present, and epithelial membrane antigen [EMA] is negative. TOP2B, MDM2, nucleolin, HIF1-alpha, and phosphorylated STAT3 were all found to be frequently expressed in subependymomas in one study of potential therapeutic targets.

**Ependymoma** - A classic ependymoma is a well-defined glioma with monomorphic cells of variable density and round to oval nuclei with speckled nuclear chromatin. Key histological findings include perivascular anucleate zones [pseudorosettes] and ependymal rosettes [true rosettes]. Myxoid degeneration, intratumoral hemorrhage, and dystrophic calcification are all characteristics. GFAP immunoreactivity is typically seen in pseudorosettes and less so in other components of the tumor. S100, EMA, and vimentin are all found in tumor cells. In some cases, focal cytokeratin immunoreactivity can be seen.

**Chordoid glioma of the third ventricle** – This is a slow-growing, non-invasive glial tumor that is histologically defined by clusters and cords of epithelioid tumor cells within a variably mucinous stroma with a lymphoplasmacytic infiltrate. TTF1 is consistently expressed in these tumors, as is diffuse GFAP reactivity. Vimentin and CD34 staining is also strong, whereas S100, EMA, and cytokeratin immunoreactivity is variable. P53 is weakly positive, while neuroendocrine markers are negative. Ki-67 proliferation is at a low level.

**Ganglioglioma** – A well-differentiated, slow-growing glioneuronal neoplasm made up of dysplastic ganglion cells and neoplastic glial cells. Eosinophilic granular bodies are more common than Rosenthal fibers. MAP2, neurofilament, chromogranin-A, and synaptophysin are all neuronal components of the tumor.

There is no specific marker that distinguishes dysplastic neurons from normal counterparts; however, chromogranin-A expression in normal neurons is usually very weak or absent, whereas diffuse and strong expression indicates a dysplastic neuron.

**Pineocytoma** – A well-differentiated, moderately cellular neoplasm composed of small, uniform, mature cells that resemble pinealocytes. It grows mostly in sheets and frequently has large pineocytomatous rosettes. The nuclei are round to oval in shape, with inconspicuous nucleoli and finely dispersed chromatin, and they lack mitotic figures. Pineocytoma cells typically express high levels of immunoreactivity for synaptophysin, neuron-specific enolase [NSE] and neurofilament. The Ki-67 proliferation index is 1%.

**Medulloblastoma** - An embryonal neuroepithelial tumor that develops in the cerebellum or dorsal brain stem and is characterized by densely packed small round undifferentiated cells with mild to moderate nuclear pleomorphism and a high mitotic count. Medulloblastoma correspond histologically to WHO grade IV regardless of their histological or genetic characterization. Medulloblastoma subtypes have been classified into four major groups based on their genetic and histological profiles: WNT-activated medulloblastomas, SHH-activated medulloblastomas, Group 3 medulloblastomas, and Group 4 medulloblastomas are the four types of medulloblastomas.[27]

**Schwannoma** - A neoplastic Schwann cell tumor composed entirely of Schwann cells that exhibit two basic architectural patterns in varying proportions, areas of compact, elongated cells with occasional nuclear palisading [Antoni A pattern] and less cellular, loosely textured cells with indistinct processes and variable lipidization [Antoni B pattern]. Tendency to show nuclear palisades which are referred as Verocay bodies.

They are S100, vimentin, calretinin, and Cd56 immunoreactive, with EMA highlighting the peripheral fibroblasts.

**Malignant peripheral nerve sheath tumor [MPNST]** – A malignant tumor characterized by tightly packed spindle cells and variable amounts of eosinophilic cytoplasm. Nuclei are typically elongated,

wavy, and tapered at the ends. It has a herringbone [fibrosarcoma-like] or interwoven fasciculated growth pattern. More than half of these cases have necrosis and mitosis greater than 4/HPF.

S100 positivity is present in 50-70 % of MPNST's, but reactivity varies by grade. It is widespread in low-grade tumors and sparse or patchy in high-grade tumors. Immunostaining for p53 protein is positive in 75% of MPNST's.

**Meningioma** – The term 'Meningioma' was coined by Harvey Cushingin the year 1922.[46] It's a group of mostly benign, slow-growing neoplasms that most likely arise from the arachnoid layer's meningothelial cells. Meningioma can have a variety of histological appearances. Meningothelial, fibrous, and transitional meningiomas are the most common subtypes.[27]

The majority have EMA immunoreactivity, though this is less consistent in atypical and malignant variants. Vimentin positivity is present in all meningiomas, but it is relatively non specific. Fibrous meningioma is the most common type of meningioma with S100 protein positivity. The mitotic index and the Ki-67 proliferation index approximately correlate with the rate of volume growth. Atypical meningioma has a Ki-67 proliferation index greater than 4%, while anaplastic meningioma has a Ki-67 proliferation index greater than 20%.

**Lymphoproliferative disorders** - Patients with acute leukemias, especially lymphoblastic leukemias, have diffuse leptomeningeal infiltration as the dominant pattern of CNS involvement. The term primary central nervous system lymphoma [PCNSL] refers to malignant lymphoid neoplasms that present in the brain, spinal cord, or meninges. Although it is not always visible, a vasocentric growth pattern with tumoral infiltration of blood vessel walls is seen in conjunction with reticulin deposition in concentric, ring-like patterns.

**Role of immunohistochemistry in diagnosis and grading of brain tumors**

Histological diagnosis of brain tumors is not easy due to overlap in morphological features among different categories, divergent differentiation within the same tumor and non-neoplastic lesion can also mimic tumor.[45] As a result, in today's world, IHC is required for accurate diagnosis and subtyping of various CNS lesions.

Glioma is a broad term that encompasses glial tumors such as astrocytoma, glioblastoma, ependymoma, oligodendroglioma, and their subtypes. Gliomas are typically GFAP positive and deficient in cytokeratin [CK], collagen, reticulin, and fibronectin. As a result, GFAP denotes their astrocytic nature. Low-grade gliomas are more likely to have BRAF gene mutations than IDH mutations.

According to WHO, diffuse gliomas are classified based on not only their growth pattern and behavior, but also on their shared IDH1 and IDH2 genetic status.[27] IDH genes frequently mutate in diffuse gliomas in younger patients [30 to 40 years]. IDH mutations are found in approximately 70% of intermediate grade [II and III] astrocytomas, more than 90% of oligodendrogliomas, and 10% of glioblastomas.

ATRX is an essential astrocytic marker. The absence of ATRX in conjunction with a TP53 mutation strongly suggests that the tumor is astrocytic.

The discovery of a single mitosis is insufficient for the diagnosis of a WHO grade III tumor. The determination of the Ki-67 proliferation index may aid in the differentiation of grade II tumors from grade III. It is used as a prognostic indicator. Many studies have shown that an increase in the Ki-67 proliferation index significantly reduces the patient's overall survival rate.

Grade IV astrocytomas are GFAP positive and often express S100, Nestin, and P53. The Ki67 index is also significantly elevated. P53 expression is found in 21-50 % of glioblastomas, while EGFR expression is found in 40-98 % of glioblastomas and correlates with the presence of gene amplification. One-tenth of all adult glioblastomas are

secondary glioblastomas, which develop in younger patients from lower grade gliomas. They usually have mutations in the genes IDH, ATRX, and TP53. They have a higher survival rate than primary glioblastomas.

There has yet to be identified a single immunohistochemical marker that is specific for oligodendroglial tumor cells. The majority of oligodendrogliomas are IDH mutated.[47] A lack of IDH1 immunopositivity, on the other hand, does not rule out oligodendroglioma.[27] IDH-mutant and 1p/19q-codeleted oligodendrogliomas, unlike most IDH-mutant diffuse astrocytomas, usually retain nuclear ATRX expression. They do not have p53 expression. Mitotic activity in WHO grade II oligodendrogliomas is low or absent. As a result, the Ki-67 proliferation index is typically 5%.

The best immunohistochemistry marker for ependymoma is epithelial membrane antigen [EMA]. It is however not always conclusively diagnostic. GFAP is typically found in pseudorosettes and in lower concentrations in other tumor components.[27]

When it comes to subependymoma, GFAP is usually present and EMA is negative. Also present are OP2B, MDM2, nucleolin, HIF1-alpha, and phosphorylated STAT3.

Neuronal brain tumors have a better patient prognosis than glial brain tumors. NeuN, synaptophysin, and neurofilaments are important markers of neuronal tumors. Because many neoplastic cells, particularly those of glioblastoma, melanoma, carcinoma, and astrocytoma, resemble neurons due to their large size or prominent nucleoli, it is critical to confirm that neurons are abnormal or neoplastic.

Medulloblastomas are multi-potential tumors that can express almost any neuro-ectodermal markers, but they specifically express synaptophysin. Non-specific neural markers like NCAM1, MAP2, and neuron-specific enolase are also expressed in varying amounts. All medulloblastoma types retain nuclear SMARCB1 and SMARCA4 expression. The absence of one of these SWI/SNF complex proteins is indicative of an atypical teratoid/rhabdoidtumor.

Schwannoma resembles fibrous meningioma, tanycytic ependymoma, subependymoma, and astrocytoma histologically. The presence of parenchymal reticulin, which is positive for type IV collagen, distinguishes schwannoma from these tumors. Also found in the nuclei of nerve sheath cells and melanocytes is SOX10, a neural crest transcription factor. This marker is found in the nuclei of schwannomas and neurofibromas. They are also positive for the Leu-7 and S100 proteins.

EMA is found in meningiomas, but it is often expressed focally and can be missed in small specimens. Meningiomas are distinguished from gliomas by the presence of EMA and the absence of GFAP. The most important IHC marker of meningioma is EMA positivity. Vimentin positivity is present in all meningiomas, but it is relatively unspecific.

A common complication of systemic cancer is secondary involvement of the CNS via direct extension or hematogenous spread.[48] Brain metastasis are most commonly seen after fifth decade of life[49] and caused by lung and breast carcinomas, followed by malignant melanomas, renal carcinomas, and colorectal adenocarcinomas.[50,51] As a result, when there is a suspicion of metastasis, IHC plays a critical role, and appropriate markers are used to make a diagnosis.

Various studies have been done to correlate the histopathology, imaging findings and role of IHC in the final diagnosis of ICSOL's.

In 2010, Parsons et al. performed an integrated genomic analysis on human glioblastoma multiforme. To identify the genetic alterations, the study included 22 cases of GBM. They sequenced 20,661 protein-coding genes, used high-density oligonucleotide arrays to detect amplifications and deletions, and used next-generation sequencing technology to analyse gene expression. In 12% of GBM patients, recurrent mutations in the active site of isocitrate dehydrogenase 1 [IDH1] were found. Mutations in IDH1 were found in a large proportion of young patients and in the majority of secondary GBM patients, and they were associated with an increase in overall survival. The study

demonstrated the value of genomic analyses in the characterization of human brain cancer and identified a potentially useful genetic change for GBM classification and targeted therapy. [52]

K Abul-Kasim et al. conducted a retrospective study in 2013 on 44 patients with suspected brain tumors, 27 [61%] males and 17 [39%] females, with a mean age of 58 [SD 17] years to assess the contribution of multimodal MRI in diagnosing ISCOL's. The concordance between the diagnosis provided by evaluating images from multimodal MRI and the final diagnosis was nearly perfect [value 0.92; 95% confidence interval 0.82 - 1], whereas the concordance between the diagnosis provided by conventional MRI and the final diagnosis was only fair [value 0.38; 95% confidence interval 0.20 - 0.56]. As a result, in the diagnosis of brain lesions, MRI was found to be superior to contrast enhanced CT. [53]

M. Tesfay et al. [2013] conducted a retrospective study on 96 patients who underwent ICSOL surgery and were followed for three years. All of the patients met the criteria, which included having CT/MRI reports and having histopathology results confirmed. Among the 96 patients, 82 had biopsy-proven diagnoses, while the remaining 14 had cysts both histopathologically and intraoperatively. Histopathology confirmed intracranial masses were meningioma 32 [39%], glioma 15 [18.3%], pituitary adenoma 14 [17%], and tuberculoma 6 [6%], [7.3%]. The sensitivity, specificity, and accuracy of CT scans in differentiating meningiomas from other intracranial masses, using the first differential as the most likely diagnosis, were 80%, 95%, and 88.6%, respectively, whereas gliomas were 71%, 85.7%, and 83%. When it came to benign masses, CT scans were more accurate, sensitive, and specific than when it came to malignant masses.[54]

According to a 2015 study by Madabhushi et al, 118 brain tumors were diagnosed based on histological examination of H&E stained sections of neurosurgical specimens. IHC was performed on all of the cases.

Astrocytomas were the most common type of tumor, accounting for 37.29% of all cases, followed by nerve sheath tumors [13.56%], meningothelial tumors 13.56%], and metastatic deposits [13.56%] [12.71%]. Pituitary and embryonal tumors were found in 6.78% and 5.08% of cases, respectively. Rarely, ependymal and oligodendroglial neoplasms, mesenchymal tumors, and malignant lymphomas were found. The study found that while histopathologic examination was the mainstay in the diagnosis and grading of the majority of brain tumors, IHC played an important supplementary role in resolving diagnostic quandaries in the routine practice of neurosurgical pathology.[55]

Nibhoria et al. conducted a study in a single tertiary care centre in 2015 for patients with clinically diagnosed CNS lesions. Overall, neuroepithelial tissue tumors [51.7%] were the most common entity, followed by meningeal tumors [34.8%], metastatic tumors [5.6%], peripheral nerve tumors [4.5%], sellar region tumors [2.3%], lymphomas [2.3%] and hematopoietic neoplasms [1.1%]. In addition, rising global trends in the incidence of CNS tumors, regardless of age, have been observed. The study highlighted the histological diversity in CNS tumors in both adults and children.[56]

Joshi H et al. conducted a histopathological correlation with radiological analysis of brain tumors in 2015, which included 96 CNS biopsies. The neoplasms accounted for 62 [64.6%] of the cases, with 60 [96.8%] being primary, 1 [1.6%] metastatic, and 1 miscellaneous lesion [1.6%]. Astrocytic tumors, Schwannomas, and Meningiomas were the three most common primary tumors. About 34 [35.4%] of the cases were non-neoplastic, with cystic lesions and non-specific inflammation being the two most common lesions. The study concluded that histopathological examination was the gold standard and, when combined with clinico-radiological correlation and the use of IHC, played an important role in arriving at a definitive diagnosis in neuro-pathology.[36]

A cross-sectional study of 102 cases conducted in 2015 by Dogar t., Imran a., Hasan m., et al. revealed 91 neoplastic [89%] and 11 non-neoplastic [11%] CNS lesions. Although CT scans and conventional MRI could provide a provisional diagnosis as well as localization of a CNS lesion, their accuracy was less than ideal. Gliomas were the most common neoplasms in this study, accounting for 47 cases [46%], followed by 13 cases [13%] of meningiomas, 4 cases of tuberculosis, and 2 cases of fungal infection, all of which presented as space-occupying lesions of the CNS when viewed with imaging techniques. Histopathology, particularly special stains like ZN, PAS, and GMS, was used to make the final diagnosis. In this study, the authors concluded that histopathological diagnosis was superior to radiological diagnosis in the context of SOLs of the brain.[57]

Kasa Lakshmi and colegues in 2015 did a study of spectrum of the lesions of central nervous system in a tertiary care hospital that included 72 neoplastic lesions of CNS, majority were astrocytomas (30) accounting 41.66%, followed by meningiomas constituting 27.77%. Out of 72 CNS tumors, 66 were primary 4 were metastatic. Among 4 metastatic tumors, the most common histological type was adenocarcinoma. There were 42 cases (58.33 %) supratentorial lesions and 22 (30.55 %) infratentorial lesions among a total of 72 major CNS neoplastic lesions. There were 6(8.33%) lesions situated in spinal cord region. Three of the spinal cord lesions were meningiomas, one was a schwannoma, and two were neurofibromas. Pituitary adenomas were the cause of the two supra sellar lesions.The study highlighted the histological diversity in CNS tumors.[58]

Hema Neelakantaiah Anadure et al. conducted a retrospective and prospective descriptive study in 2016 that included 62 cases of CNS lesions, of which 12 [19.4%] were non-neoplastic, 6 [50%] were cystic lesions, and 4 [33.4%] were cerebral abscesses. There were 50 [80.6%] cases of neoplastic lesions, with 48 [96%] being primary and 2 [4%] being metastatic. Gliomas were the most common type of primary

tumor, accounting for 24 [50%] of all cases, with 16.7% being Glioblastoma Multiforme [GBM] and pilocyticastrocytomas, respectively, followed by schwannomas [14%] and meningothelial tumors [12%]. The importance of IHC in histopathology was emphasised. The availability of clinical information and neuro imaging techniques such as CT scan and MRI were also critical. [59]

Jat KC et al. screened 59 patients with CNS lesions in 2016 and analysed their histopathological profiles. The ages of the patients ranged from 3.5 to 65 years, with a mean of 41.2 years and a male to female ratio of 1.8:1. They discovered that the frontal lobe was the most affected region [37.3%]. The majority of tumors [67.8%] originated from neuroepithelium, followed by meninges [22.03%]. Astrocytoma was the most common type of CNS tumor in this study, followed by meningioma. Males had a much higher risk of developing a CNS tumor than females [1.8:1]. As a result, precise histological diagnosis of CNS tumors was required to predict prognostic factors.[60]

Jindal Neha et al. studied 80 patients with ICSOL's using CT and MRI in 2016. Imaging findings were compared to clinical and histopathological findings [wherever available]. The majority of patients were between the ages of 31 and 50 [mean age 42.2years]. The male-to-female ratio was 3:2. Solitary lesions were present in 58 [72.5%] of the patients, while multiple lesions were present in 22 [27.5%]. 76.2% of lesions were supratentorial, while 23.7% were infratentorial. 76.2% of the patients had neoplastic lesions, while 23.7% had non-neoplastic lesions. Metastases were the most common neoplastic lesion, while arachnoid cysts were the most common non-neoplastic lesions. The imaging sensitivity for neoplastic lesions was 90%, the specificity was 57%, the accuracy was 84.2%, the positive predictive value [PPV] was 90%, and the negative predictive value [NPV] was 57%. While imaging sensitivity for non-neoplastic lesions was 57%, specificity was 90%. The accuracy was 84.2%, the PPV was 57%, and the NPV was 90%. The study concluded that these modalities aided in the early diagnosis

and localization of the SOL, and that when combined with advanced neurosurgical techniques, they improved the prognosis of mass lesions.[61]

Adnan H.A., Kambhoh U.A., Majeed S., et al. investigated 185 intracranial lesions in 2017. The diagnosis was made on routinely processed tissue that had been stained with H&E. Out of the 185 CNS lesions studied, 107 [58%] were males and 78 [42%] were females. The case had a minimum age of 8 years and a maximum age of 75 years. The most common lesions were of a neoplastic nature, with glioma being the most common [35%], followed by meningioma [23%]. Fungal infections, pituitary adenoma, schwannoma, neurofibroma, carotid body paraganglioma, craniopharyngioma, arachnoid cyst, medulloblastoma, and medulloepithelioma were among the other lesions. The histological spectrum of non-neoplastic and neoplastic CNS lesions is described in this study.[32]

Similarly, in a 2017 prospective study conducted by Renu Thambi et al, 510 cases of brain tumors were studied over a seven-year period, and the analysis revealed that the majority of the brain tumors occurred between the ages of 40 and 60 years, with a male to female ratio of 0.9:1. Except for the occipital lobe, the majority of cases involved the dura and cerebral lobes, with meningioma and glial tumors being the most common broad histological types. Males had a higher rate of WHO grade IV tumors and metastasis than females.[62]

Another study, conducted in 2017, by B.N. Kumarguru et al, examined various histopathological patterns of CNS lesions and evaluated the association with the distribution of ABO blood groups in comparison to a control group. The majority of the 147 cases included in the study were neoplastic lesions [84.35%], with neuroepithelial tumors [29.25%] being the most common pattern. Non-neoplastic lesions made up only 15.64% of the total, with inflammatory lesions [8.16%] being the most common pattern. ABO blood group information was available in 92

[84.4%] of the neoplastic lesions studied, including 71 [48.29%] of primary CNS neoplasms. The most common blood group in neoplastic lesions was group O [40.21%]. The association between CNS neoplasms and ABO blood groups was not statistically significant, but there was a distinct change in the pattern of ABO blood group distribution between neoplastic lesions and control groups.[29]

Sunila et al. evaluated the histology of 88 ICSOL cases in 2018. Out of 88 cases, the majority 80 cases [90.9%] were neoplastic lesions, while the remaining 8 cases [9.09%] were non-neoplastic lesions. Complete concordance was observed in cases where the radiological diagnosis matched the final histopathological diagnosis exactly. Partial concordance was seen in cases where the radiological diagnosis differed slightly from the final histopathological diagnosis. As a result, radiological examination was the only option for preoperative assessment, and histopathology was regarded as the gold standard for evaluating CNS lesions.[1]

Hamdani et al. conducted a study on the histopathological spectrum of brain tumors in 2019 that included 117 retrospective cases of intracranial lesions. A diverse histopathological spectrum of CNS tumors was observed and classified using the WHO classification system of 2007. The most common lesion was meningioma 48 cases [41.02%], followed by astrocytoma 41 cases [35.04%]. Grade I lesions were the most common with 39 cases among the 48 cases of meningiomas, followed by Grade III lesions [7 cases] and Grade II lesions [2 cases]. The most common grade of astrocytoma was Grade IV [21 cases], followed by Grade II [14 cases], Grade I [2 cases], and Grade III [1 case]. Brain tumors are diverse, with differences not only in histomorphological features but also in biological background and disease course. Despite the availability of advanced imaging techniques, histopathological examination remains the gold standard in their diagnosis, and IHC also plays a significant role in differential diagnosis and improving diagnostic accuracy.[63]

Meena B. Patil and colleagues conducted a 5-year retrospective study on 124 ICSOL biopsies in 2019. In 70 cases, intraoperative diagnosis was desired, and the final diagnoses were made by routine histopathology in all cases. Out of 124 cases clinically diagnosed as ICSOLs, 104 were neoplastic lesions and 20 were non-neoplastic lesions. There was a slight female predominance in lesions [males – 56 and females – 68]. Males were more likely to develop astrocytoma, whereas females were more likely to develop meningioma. The male to female ratio for craniopharyngioma, pituicytoma, medulloblastoma, and primitive neuroectodermal tumor was 1:1. The study emphasized the histological diversity in CNS tumors in both adult and pediatric age groups, emphasizing the significance of histopathological features.[64]

In 2019, Bhatt et al. conducted a study to assess the pathologic spectrum of non-neoplastic cystic lesions in the CNS [both developmental and acquired] and to highlight the role of histopathology in the diagnosis of these cystic lesions. The study included 538 patients ranging in age from 5 months to 90 years [M:F:1:1.05]. Non-infective cysts [489/538, 90.8%] outnumbered infective cysts [49/539, 9.2%], with epidermoid cysts [132/538, 24.5%] being the most common, followed by colloid cysts [126/538, 23.4%] and arachnoid cysts [111/538, 20.6%]. Neurocysticercosis was the most common infective cyst [42/538, 7.8%], followed by hydatid cyst [7/538, 1.3%]. Intracranial cysts were more common [415/538, 77.1%] than spinal cysts [123/538, 22.9%]. Because there are overlapping clinical features, image findings, and lining in a variety of cystic lesions in the CNS, histological analysis is critical in the evaluation of these lesions.[65]

A prospective study published in 2019 by H. Beuli et al. included 41 consecutive patients with intracranial meningioma which were surgically treated, with 17 [41.57%] males and 24 [58.53%] females. Prior to surgery, all patients had 1.5T MRIs. On all cases, histopathology and the Ki-67 index were performed. Ki-67 was found to

be statistically significant when compared to pial invasion and bone infiltration.[66]

Khonglah et al. studied the histological spectrum of central nervous system lesions in 2020, including 238 cases of ICSOL's. There were 205 neoplastic cases [86.13%] and 33 non-neoplastic cases [13.86%] among the 238 cases. The average age was 36 years [range: 23 days to 80 years]. The peak incidence occurred between the ages of 31 and 40, accounting for 52 [21.84%] of the cases. Males made up 121 [50.84%] of the study population, while females made up 117 [49.15%], for a male to female ratio of 1.03:1. The study included 48 [23.41%] cases of pediatric brain tumors [age 18 years]. Out of 205 [86.13%] neoplastic lesions encountered, 200 [97.56%] were primary lesions and 5 [2.43%] were metastatic lesions. Gliomas [27.80%] were the most common type of primary tumor, followed by meningeal tumors [20.97%] and tumors of the cranial and paraspinal nerves [18.04%]. The research emphasizes the histological diversity of CNS lesions. Although modern imaging techniques can provide a provisional diagnosis for these diseases, histological examination with further IHC use remains the gold standard in the diagnosis and grading of all CNS lesions. [67]

Thus, as per above literature, histopathology remains the gold standard for diagnosis of ICSOL's. Imaging studies are helpful to seen the location and extent of the lesions. Use of IHC biomarkers also shows a great importance in accurate diagnosis and grading of many tumors. Hence the present study was undertaken to examine the histopathologic spectrum of ICSOL's and to correlate them with IHC wherever possible.

# MATERIAL AND METHODS

The present study was a prospective and retrospective study on histopathology of intracranial lesions of specimens received in the Department of Pathology, Dayanand Medical College & Hospital, Ludhiana over a period of 5 years (3.5 years retrospectively from 1st July 2016 to 31st December 2019 and 1.5 years prospectively from 1st January 2020 to 30th June 2021). During this period all the specimens were analyzed with respect to light microscopic and IHC findings (wherever possible). Total of 71,244 specimens were received in the Histopathology department, out of which 266 were intracranial lesions. These specimens were classified accordingly after light microscopy and using immunohistochemistry wherever possible.

**METHOD OF COLLECTION OF DATA**

**Inclusion criteria**: All the biopsies from cranial vault.

**Exclusion criteria**: All biopsies of cranial nerves, peripheral nerves and spinal cord**.**

**Duration of study**: Retrospective 3 ½ years and prospective 1 ½ years.

**Study Design**: This is prospective and retrospective study. **Methodology**: Biopsies were received in containers containing 10% formalin. Random representative sections were taken from the specimen and then were processed. Paraffin embedding and subsequent staining by Hematoxylin and Eosin was done. Special stains like PAS, AFB, GMS, Reticulin were done wherever required. IHC for GFAP, IDH, ATRX, P53, MIB-1, CK, LCA, EMA, synaptophysin etc. were done wherever needed.

**Paraffin Block Tissue Processing**

The tissue fixed in 10 % formalin was processed through a series of solutions in the automatic tissue processor as follows:

- Acid formalin for 4 hours
- Formal alcohol for 1 hour
- 80% ethyl alcohol for 1 hour

- 90% ethyl alcohol for 1 hour
- Absolute ethyl alcohol for 1 hour
- Acetone [1st change ] for 1 hour
- Acetone [2nd change] for 1 hour
- Xylene [1st change ] for 1 hour
- Xylene [2nd change] for 1 hour
- Paraffin [1st change at 58-69°C] for 1 hour
- Paraffin [2nd change at 58-69°C] for 1 hour

The paraffin embedded tissue blocks were cut at 2-3um thickness by rotary microtome. The sections were then transferred onto albunimized slides. These sections were labelled properly with the help of glass marking pencil.

**Procedure of Hematoxylin and Eosin staining**:

- Slides were kept on hot plate in order to dewax the sections at temperature upto 60ºC.
- Then the slides were deparaffinised in xylene
- Then hydration was done by passing through descending rates of alcohol to water
- The slides were then stained in Harris Hematoxylin for 15 minutes. Sections were washed well in running tap water until sections turned blue. These sections were then differentiated in 1 % acid alcohol [1% HCL in 70% alcohol] for 5-10 seconds, washed well in tap water till sections were again blue for 5 minutes or less.
- These were again stained in 1% Eosin for 10 minutes followed by washing in running tap water for 1-5 minutes.
- Sections were dehydrated once again through ascending grades of alcohol, cleaned in xylene and mounted with DPX [Distyrenedibutylphtalate xylene] avoiding air bubbles.
- Stained slides were then subjected to microscopic examinations under both [10X] and high power [40X]

# OBSERVATIONS & RESULTS

The present 5 year study, 3.5 years retrospective [1st July 2016 to 31st December 2019] and 1.5 years prospective [1st January 2020 to 30th June 2021] on histopathological spectrum of intracranial lesions was conducted in the department of pathology, Dayanand Medical College & Hospital, Ludhiana.

During the study period a total of 71,244 specimens were received in the department, out of which 266 were intracranial lesions constituting 0.37%. In all cases relevant clinical history, clinical findings and radiological findings were noted and were confirmed by histological analysis with use of special stains and IHC wherever possible. 79.70% of probable diagnosis made by radiological findings were accurate on histological analysis. These intracranial lesions were classified into non-neoplastic and neoplastic lesions. There were 220 neoplastic and 46 non-neoplastic lesions.

**Table 1: Distribution of Intracranial space occupying lesions [ICSOL's] [n=266]**

| Type | Number of cases | %age |
|---|---|---|
| Non-neoplastic | 46 | 17.29 |
| Neoplastic | 220 | 82.71 |
| Total | 266 | 100 |

**NON-NEOPLASTIC LESIONS**

The non-neoplastic lesions included 11 cases each of arteriovenous malformations and epidural/subdural hematoma. There were 5 cases of intraparenchymal hemorrhage, 4 cases each of pachymeningitis and abscess [non-specific], 3 cases of tuberculosis, 2 cases of brain infarct, and 1 case each of arachnoid cyst, epidermal inclusion cyst, aspergillosis, amoebic brain abscess and aneurysm.

**Table 2: Histological distribution of non-neoplastic lesions [n=46]**

| Type | Number of cases | %age |
|---|---|---|
| Arteriovenous malformations | 11 | 23.91 |
| Epidural/subdural hematoma | 11 | 23.91 |
| Intrparenchymal hemorrhage | 5 | 10.86 |
| Pachymeningitis | 4 | 8.69 |
| Abscess [non-specific] | 4 | 8.69 |
| Tuberculosis | 3 | 6.52 |
| Infarct | 2 | 4.34 |
| Subdural hemorrhage | 1 | 2.18 |
| Arachnoid cyst | 1 | 2.18 |
| Epidermal inclusion cyst | 1 | 2.18 |
| Amoebic abscess | 1 | 2.18 |
| Aspergillosis | 1 | 2.18 |
| Aneurysm | 1 | 2.18 |
| Total | 46 | 100 |

Arteriovenous malformation and blood clot were the most commonly occurring non-neoplastic ICSOL's in our study, both had 11 cases [23.91%] each. Arteriovenous malformation showed numerous thick and thin walled, dilated and congested blood vessels having variable wall thickness. These were seen below 50 years of age in our study.

5 out of 11 cases diagnosed as epidural/subdural hematomahad history of head trauma. Intrparenchymal hemorrhage had 5 cases [10.86%] and showed areas of intrparenchymal hemorrhage with many dilated and congested blood vessels.

4 cases [8.69%] of pachymeningitis showed infiltration of dura mater by inflammatory cells seen mainly as neutrophils and few lymphocytes along with areas of necrosis and sheets of hemosiderin laden macrophages.

4 cases diagnosed as abscess, non-specific showed areas of necrosis having sheets of inflammatory cells mainly neutrophils admixed with foamy macrophages. Special stains for AFB and fungus were done in all cases and came out to be negative. 2 cases had preceding history of chronic suppurative otitis media [CSOM].

One case of amoebic abscess showed extensive areas of necrosis with dense inflammatory infiltrate along with trophozoites of Entamoeba histolytica, which were highlighted on PAS stain.

One case of arachnoid cyst showed a cyst wall having lining of mengiothelial cells. Cyst wall also showed focal infiltration by chronic inflammatory infiltrate.

A solitary case of epidermal inclusion cyst showed a cyst wall lined by keratinized stratified squamous epithelium. One case of aspergillosis showed infiltration of brain parenchyma by neutrophils with tiny foci of necrosis. Within the foci of necrosis were seen septate and acute angle branching fungal hyphae of aspergillus which were confirmed with PAS and GMS stain.

**Table 3: Age wise distribution of non-neoplastic lesions [n=46]**

| Age [years] | Number of cases | %age |
|---|---|---|
| 1-10 | 2 | 4.34 |
| 11-20 | 3 | 6.52 |
| 21-30 | 4 | 8.70 |
| 31-40 | 6 | 13.05 |
| 41-50 | 8 | 17.40 |
| 51-60 | 15 | 32.60 |
| 61-70 | 5 | 10.87 |
| 71-80 | 3 | 6.52 |
| Total | 46 | 100 |

The age group analysis of non-neoplastic lesions showed that the older age group of 51-60 years had the highest predilection of occurrence of non-neoplastic lesions. Mean age for non-neoplastic lesions was 46.4 years [youngest - 2 years & oldest - 80 years].

**Table 4: Gender wise distribution of non-neoplastic lesions [n=46]**

| Gender | Number of cases | %age |
|---|---|---|
| Male | 33 | 71.73 |
| Female | 13 | 28.27 |
| Total | 46 | 100 |

The gender wise analysis of non-neoplastic lesions revealed that there was a definite male preponderance with 33 cases [71.73%]. The male: female ratio was 2.5:1.

**Table 5: Distribution of laterality of non-neoplastic lesions. [n=46]**

| Location | Number of cases | %age |
|---|---|---|
| Right | 29 | 63.04 |
| Left | 17 | 36.96 |
| Total | 46 | 100 |

Right side [29 cases] of brain was more commonly affected by the non-neoplastic lesions than the left side [17 cases].

**Table 6: Distribution of site of non-neoplastic lesions. [n=46]**

| Site | Number of cases | %age |
|---|---|---|
| Temporal | 18 | 39.20 |
| Frontal | 14 | 30.50 |
| Parietal | 6 | 13.0 |
| Temporo-parietal | 4 | 8.60 |
| Parieto-occipital | 2 | 4.35 |
| Fronto-parietal | 2 | 4.35 |
| Total | 46 | 100 |

The commonest intracranial site for non-neoplastic lesions came out to be temporal lobe with 18 cases [39.20%] and arteriovenous malformation was the most common lesion [5 cases] followed by epidural/subdural hematoma [4 cases] in temporal lobe. Frontal lobe was second most common site with 14 cases [13.04%], followed by parietal lobe, having 6 cases [8.70%]. The least common site for non-neoplastic lesions was parieto-occipital and fronto-parietal with 2 cases [4.35%] each.

**NEOPLASTIC LESIONS:**

The neoplastic ICSOL's were divided into primary and secondary brain tumors. There were 203 cases [92.28%] of primary and 17 cases [7.72%] of secondary brain tumors.

**Table 7: Types of neoplastic ICSOL's [n=220]**

| Types | Number of cases | %age |
|---|---|---|
| Primary | 203 | 92.28 |
| Secondary | 17 | 7.72 |
| Total | 220 | 100 |

**Table 8: Histological distribution of primary brain tumors [n=203]**

| Types | Number of cases | %age |
|---|---|---|
| Diffuse astrocytoma | 9 | 4.43 |
| Anaplastic astrocytoma | 5 | 2.46 |
| Glioblastoma | 60 | 29.6 |
| Oligodendroglioma | 15 | 7.40 |
| Anaplastic oligodendroglioma | 3 | 1.48 |
| Anaplastic oligoastrocytoma | 2 | 0.99 |
| Pilocytic astrocytoma | 4 | 1.97 |
| Pleomorphic xanthoastrocytoma | 1 | 0.49 |
| Ependymoma | 2 | 0.99 |
| Anaplastic ependymoma | 2 | 0.99 |
| Astroblastoma | 1 | 0.49 |
| Ganglioglioma | 1 | 0.49 |
| Medulloblastoma | 3 | 1.48 |
| Schwannoma | 8 | 3.94 |
| Meningioma | 52 | 25.60 |
| Haemangiopericytoma | 1 | 0.49 |
| Haemangioblastoma | 2 | 0.99 |
| Primitive neuroectodermaltumor | 1 | 0.49 |
| Lymphoma | 8 | 3.94 |

| | | |
|---|---|---|
| Craniopharyngioma | 1 | 0.49 |
| Pituitary adenoma | 22 | 10.80 |
| Total | 203 | 100 |

In present study glioblastoma, was found to be the commonest primary brain tumor with 60 cases [29.6%], followed by the meningiomas having 52 cases [25.50%] and pituitary adenomas with 22 cases [10.80%]. Pleomorphic xanthoastrocytoma, astroblastoma, ganglioglioma, haemangioblastoma, primitive neuroectodermal tumor and craniopharyngioma were the least common tumors with one case each [0.49%].

All the tumors were classified according to WHO classification of tumors of the central nervous system 2016.

**Astrocytic Tumors**

**Table 9: Histological distribution of astrocytic tumors [n=74]**

| Types | Number of cases | %age |
|---|---|---|
| Diffuse astrocytoma | 9 | 12.2 |
| Anaplastic astrocytoma | 5 | 6.76 |
| Glioblastoma | 54 | 72.93 |
| Giant cell glioblastoma | 2 | 2.70 |
| Gliosarcoma | 4 | 5.41 |
| Total | 74 | 100 |

Regarding distribution of astrocytic tumors the major bulk was formed by the glioblastomas with 54 cases [72.93%], followed by diffuse astrocytomas [9 cases] [12.2%] and anaplastic astrocytoma [5 cases] [6.76%].

All the cases of glioblastomas showed marked increase in cellularity, pleomorphism, nuclear hyperchromasia and brisk mitotic activity. Out

of 54 cases of glioblastoma, 41 [77.35%] cases showed prominent microvascular proliferation and 19 [35.18%] cases showed geographical necrosis palisaded by tumor cells.

IHC was done in 10 cases of glioblastoma, out of which 9 cases [90%] were IDH-1 wildtype and 1 case showed IDH-1 mutation [10%]. GFAP was consistently positive in tumor cells in all the 10 cases, ATRX showed positivity in 9 cases and P53 done in 8 cases showed immunopositivity in 6 cases. Ki67 proliferation index ranged from 15 to 50%.

4 cases [3.84%] of gliosarcoma showed a biphasic pattern exhibiting alternating areas of glial and mesenchymal differentiation. Mesenchymal spindle cell areas were highlighted on reticulin stain and were negative for GFAP. IHC revealed positivity for vimentin and ATRX. IDH-1 was negative [wild type] in all cases.

2 cases [1.92%] showed numerous multinucleated giant cells along with cellular areas having pleomorphic tumor cells and brisk mitotic activity. They were diagnosed as giant cell glioblastoma. Both the cases were IDH-1 wildtype and were positive for GFAP and P53. Ki67 proliferation index was 20% and 70% respectively.

Diffuse astrocytomas constituted 9 cases [12.2%]. All the 9 cases showed moderately increased cellularity with tumor cells arranged in sheets against a fibrillary background. Tumor cells showed nuclear atypia, hyperchromatic nuclei and moderate amount of cytoplasm. Occasional mitotic figures were noted in 3 cases. However, no significant necrosis, microvascular proliferation and brisk mitosis was observed in any of the cases. IHC was positive for GFAP in two cases, one of which was an IDH-1 mutant and the other was an IDH wildtype.

5 cases of anaplastic astrocytoma showed high cellularity with distinct nuclear atypia along with increased mitotic activity. However, no significant necrosis or microvascular proliferation was noted in any case. IHC done in 2 cases showed immunopositivity for GFAP, loss of ATRX and Ki67 proliferation index of 10% and 20% respectively. IDH-1 mutation was seen in one case and the second case was IDH wild-type.

**Table 10: Age wise distribution of astrocytic tumors [n=74]**

| Age [years] | Number of cases | %age |
|---|---|---|
| <20 | 1 | 1.35 |
| 21-30 | 5 | 6.75 |
| 31-40 | 6 | 8.11 |
| 41-50 | 14 | 18.90 |
| 51-60 | 27 | 36.49 |
| 61-70 | 19 | 25.70 |
| 71-80 | 1 | 1.35 |
| >80 | 1 | 1.35 |
| Total | 74 | 100 |

The age group analysis of astrocytic tumors showed that age group of 51-60 years showed the highest tendency of occurrence [27 cases, 36.49%]. This was followed by the age group of 61-70 years with 19 cases [25.70%]. So the overall occurrence of astrocytic tumors was highest in the adult age [51-70years] accounting for 62.19% cases and glioblastoma was the commonest tumor with 48 cases. The least affected population were the patients of the eighth and ninth decade.
Mean age for astrocytic tumors was 52.9 years [youngest - 6 years & oldest - 84 years].

**Table 11: Gender wise distribution of astrocytic tumors [n=74]**

| Gender | Number of cases | %age |
|---|---|---|
| Male | 53 | 71.62 |
| Female | 21 | 28.38 |
| Total | 74 | 100 |

The gender wise analysis of astrocytic tumors revealed a definite male preponderance with 53 cases [71.62%]. Male:female ratio was 2.5:1

**Table 12: Distribution of laterality of astrocytic tumors [n=74]**

| Side | Number of cases | %age |
|---|---|---|
| Right | 36 | 48.65 |
| Left | 38 | 51.35 |
| Total | 74 | 100 |

Both right and left sides were almost equally affected by the astrocytic tumors with left comprising 38 cases [51.35%] and right side 36 cases [48.65%].

**Table 13: Distribution of site of astrocytic tumors [n=74]**

| Site | Number of cases | %age |
|---|---|---|
| Frontal | 26 | 35.10 |
| Temporal | 17 | 23.0 |
| Parietal | 13 | 17.60 |
| Fronto-parietal | 6 | 8.10 |
| Temporo-parietal | 3 | 4.05 |
| Parieto-occipital | 3 | 4.05 |
| Fronto-temporal | 2 | 2.70 |
| Occipital | 2 | 2.70 |
| Cerebellum | 1 | 1.35 |
| Perisylvial | 1 | 1.35 |
| Total | 74 | 100 |

In the present study frontal lobe [26 cases] [35.10%] was the most commonly affected region followed by temporal lobe [17 cases] [23%] accounting for 35.10% & 23% cases respectively. Cerebellum and the perisylvian region were the least commonly sites in our study, each comprising one case.

**Table 14: Grade wise distribution of astrocytic tumors [n=74]**

| Grade | Frequency | %age |
|---|---|---|
| II | 9 | 12.16 |
| III | 5 | 6.76 |
| IV | 60 | 81.08 |
| Total | 74 | 100 |

The astrocytic tumors were graded according to WHO classification of CNS tumors 2016. It was observed that the grade IV tumors i.e glioblastomas were the commonest having 60 cases accounting for 81.08% of the astrocytic tumors.

**Oligodendroglial Tumors**

**Table 15: Histological distribution of oligodendroglial tumors [n=20]**

| Types | Number of cases | %age |
|---|---|---|
| Oligodendroglioma | 15 | 75 |
| Anaplastic oligodendroglioma | 3 | 15 |
| Anaplastic oligoastrocytoma | 2 | 10 |
| Total | 20 | 100 |

Histological distribution of oligodendroglial tumors included 15 cases [75%] of oligodendroglioma, 3 cases [15%] of anaplastic oligodendroglioma and 2 [10%] cases of anaplastic oligoastrocytoma.

Oligodendroglioma cases showed cellular areas with round enlarged nuclei, abundant amount of cytoplasm & perinuclear halo. Out of 15 cases 10 [66.66%] showed prominent chicken wire vasculature, 5 [35.71%] showed microcalcifications and 5 [33.33%] showed perineuralsatellitosis. None of the cases showed significant mitosis or microvascular proliferation.

IHC done in 1 case showed IDH-1 mutation with retained nuclear expression of ATRX. Ki67 proliferation index in 2 cases was 8% and 10% respectively.

3 cases of anaplastic oligodendroglioma showed additional features seen as increased cellularity, nuclear hyperchromasia, microvascular proliferation and increased mitotic activity [>6/10 hpf]. One case also showed areas of necrosis.

2 cases diagnosed as anaplastic oligoastrocytoma showed cellular tumor with dual cell population. Tumor cells with round nuclei, abundant clear cytoplasm with perinuclear halo were present against chicken wire vasculature representing oligodendrogial component. Also seen were mildly pleomorphic, hyperchromatic tumor cells in diffuse sheets against fibrillary background - astrocytic component. Both the case showed focal cystic change, prominent mitotic activity and focal microvascular proliferation.

**Table 16: Age wise distribution of oligodendroglial tumors [n=20]**

| Age [years] | Number of cases | %age |
|---|---|---|
| 21-30 | 4 | 20 |
| 31-40 | 6 | 30 |
| 41-50 | 4 | 20 |
| 51-60 | 5 | 25 |
| >60 | 1 | 5 |
| Total | 20 | 100 |

Most common age bracket for oligodendroglial tumors came out to be 31- 40 years comprising 6 cases [30%] and all of these cases were of oligodendroglioma.

Mean age for oligodendroglial tumors was 41.6 years [youngest - 21 years & oldest - 62 years].

**Table 17: Gender wise distribution of oligodendroglial tumors [n=20]**

| Gender | Number of cases | %age |
|---|---|---|
| Male | 14 | 70 |
| Female | 6 | 30 |
| Total | 20 | 100 |

There was male predominance in oligodendroglial tumors. Male to female ratio came out to be 2.3:1.

**Table 18: Distribution of laterality of oligodendroglial tumors [n=20]**

| Side | Number of cases | %age |
|---|---|---|
| Right | 09 | 45 |
| Left | 11 | 55 |
| Total | 20 | 100 |

Left sided brain had higher predilection for oligodendroglial tumors with 11 cases [55%].

**Table 19: Distribution of site of oligodendroglial tumors [n=20]**

| Site | Number of cases | %age |
|---|---|---|
| Frontal | 12 | 60 |
| Fronto-temporal | 3 | 15 |
| Parieto-occipital | 3 | 15 |
| Temporal | 1 | 5 |
| Fronto-parietal | 1 | 5 |
| Total | 20 | 100 |

In our study frontal region was the commonest site for oligodenfroglial tumors with 12 cases [60%]. 10 out of 12 cases of frontal region were diagnosed as oligodendroglioma and 2 cases were of anaplastic oligoastrocytoma.

**Table 20: Grade wise distribution of oligodendroglial tumors [n=20]**

| Grade | Number of cases | %age |
|---|---|---|
| II | 15 | 75 |
| III | 5 | 25 |
| Total | 20 | 100 |

Grade II oligodendroglial tumors were more than grade III tumors, constituting 15 cases [75%] out of 20.

**Other astrocytic tumors**

Our study included 4 cases of pilocytic astrocytoma, 3 out of which were present in the posterior cranial fossa and 1 in the frontal lobe. 3 showed younger age of presentation i:e 5, 11 and 14 years and 1 case was seen in 40 years of age..

All the cases of pilocytic astrocytoma showed a biphasic pattern with hypocellular and hypercellular areas. Hypercellular areas showed cells with elongated spindle shaped nuclei against a fibrillary background. The hypocellular areas showed cystic change with cells having round nuclei. Rosthenthal fibers and eosinophilic granular bodies were seen in all the cases in variable proportions. No atypical mitosis, necrosis and microvascular proliferation were seen in any of the cases. IHC was performed one 1 case and showed immune-positivity for GFAP and ATRX. IDH-1 was negative [wild-type] and Ki-67 proliferation index was 2%.

A solitary case of pleomorphic xanthoastrocytoma was diagnosed, seen in an elderly male of 64 years. Morphology showed numerous multinucleated giant cells intermingled with cells having pleomorphic hyperchromatic nuclei present against a fibrillary background. Occasional mitotic figure and a tiny focus of necrosis was seen. IHC in this case revealed positivity for GFAP, S100 and vimentin. Synaptophysin was negative and Ki-67 proliferation index was 1%.

**Other gliomas**

A solitary case of astroblastoma was seen in a 12 year old female, who presented with right temporo-parietal SOL. Morphology showed cellular tumor with tumor cells in sheets and perivascular configuration. Cells were elongated with abundant eosinophilic cytoplasm with prominent process extending to a central blood vessel. No significant mitosis, necrosis and endothelial cell proliferation was seen.
Cytoplasmic Immunoreactivity for vimentin, S100, and GFAP with Ki-67 proliferation index of 8% was noted with IHC in this case.

**Ependymal tumors**

2 cases of ependymoma showed cellular areas with tumor cells arranged in diffuse sheets and were palisaded around vascular channels forming pseudorosettes. Tumor cells arranged around a central lumen – true rosettes were seen in 1 case. Tumor cells also had round hyperchromatic nuclei with moderate cytoplasm against fibrillary background with focal areas of necrosis. However no significant mitotic activity and microvascular proliferation was seen.

2 cases diagnosed as anaplastic ependymoma showed additional morphological features seen as high mitotic count and large areas of necrosis. Tumor cells in these cases showed marked atypia and hyperchromasia. IHC done in 1 case showed positivity for GFAP and

p53 with intact ATRX expression. Ki-67 proliferation index of 20% was noted.

2 cases of ependymoma were in fourth ventricle and presented in younger age group [3 and 16 years]. However, anaplastic ependymoma showed predilection for slightly higher age group in our study [31 and 56 years].

**Neuronal and mixed neuronal-glial tumors**

A solitary case of ganglioglioma in a 48 year old male was observed during our study period. Morphology showed a cellular tumor with tumor cells in diffuse sheets intermingled with occasional dysplastic neurons. Tumor cells had round hyperchromatic nuclei and abundant amount of clear cytoplasm with well-defined cytoplasmic boundaries. Extensive areas of calcification and occasional mitotic figure was seen. No endothelial cell proliferation was present.

**Embryonal tumors**

3 cases of medulloblastoma were observed in our study period. All 3 cases were located in the posterior fossa and presented at the age of 5, 20 and 26 years respectively. Histology in 2 cases showed a cellular tumor with tumor cells arranged in diffuse sheets, cords and perivascular configuration. Tumor cells had round to oval hyperchromatic nuclei and scant to moderate amount of cytoplasm. Numerous mitotic figures were also noted. 1 case showed nodular pale islands having reduced cellularity which were surrounded by densely packed cells having round to oval hyperchromatic nuclei and scant to moderate amount of cytoplasm. Reticulin free pale islands were noted on special stain. IHC done on this case showed immunopositivity for CD-56, Synaptophysin, NSE, CD-99 and was labelled as desmoplastic medulloblastoma.

**Tumors of the cranial and paraspinal nerves**

There were 8 cases of schwannoma in our study, 5 of these were located at cerebellopontine angle. Mean age of presentation was 44 years. Morphology showed hypercellular [Antoni-A] and hypocellular [Antoni-B] areas. Hypercellular areas showed cells having spindled vesicular nuclei with ill-defined eosinophilic cytoplasm and hypocellular areas showed cells having round to ovoid nuclei.

Tumor nuclei showed a tendency to align in alternating parallel rows, forming nuclear palisades known as Verocay body. These were seen in 3 out of 8 cases. Necrosis, nuclear atypia and mitosis were not observed in any case.

**Meningioma**

There were 52 cases of meningioma, constituting 25.60% of all primary brain tumors.

**Table 21: Histological distribution of meningiomas [n=52]**

| Type | Number of cases | %age |
|---|---|---|
| Transitional | 29 | 55.77 |
| Meningothelial | 7 | 13.46 |
| Angiomatous | 5 | 9.62 |
| Atypical | 4 | 7.70 |
| Anaplastic | 2 | 3.85 |
| Psammomatous | 1 | 1.92 |
| Fibrous | 1 | 1.92 |
| Microcystic | 1 | 1.92 |
| Metaplastic | 1 | 1.92 |
| Chordoid | 1 | 1.92 |
| Total | 52 | 100 |

Histologically transitional meningiomas formed the major bulk with 29 cases comprising of 55.77% of all meningiomas. It was followed by meningothelial, 7 cases [13.46%] and angiomatous, 5 cases [9.62%]. Transitional meningiomas showed coexistence of meningothelial and fibrous patterns with presence of whorls and fascicles. Psammoma bodies wee seen in 18 [62.06%] and intranuclear pseudo inclusions were seen focally in 4 [13.80%] cases. There was minimal nuclear pleomorphism and no sheeting pattern, small cell change was noted in any case.

The second common subtype was meningothelial meningioma, with 7 cases which were characterized by lobular and syncytial architecture of cells having round to oval nuclei, delicate chromatin, inconspicuous nucleoli and moderate amount of cytoplasm with ill-defined cytoplasmic boundaries. No nuclear atypia, mitosis, sheeting pattern, small cell change was seen any of the cases.

Angiomatous meningiomas comprised of 5 cases [9.62%]. Predominence of blood vessels in the morphology helped in making this diagnosis. Tumor cells had round to oval vesicular nuclei, inconspicuous nucleoli and moderate amount of eosinophilic cytoplasm. Psammoma bodies were seen in one case. However, no any other histological feature suggestive of higher grade was noted.

Atypical meningiomas constituted 4 cases [7.70%]. Out of 4 cases 3 showed increased cellularity with mitotic figures >4/10HPF and one case showed extensive areas of necrosis. Brain invasion was seen in 2 [50%] cases. However, no cytological/nuclear atypia was observed.
Anaplastic meningioma comprised of 2 cases [3.85%] of all meningiomas included in this study. Morphology showed tumor cells with moderate nuclear pleomorphism arranged in whorls and fascicles. Both the cases showed brisk mitosis [>20/10hpf] and foci of necrosis. Ki67 labelling index was done in one case and came out to be 25%.

Psammomatous, fibrous, microcystic, metaplastic and chordoid types were in equal proportions with one case each. Psammomatous meningioma showed predominance of psammoma bodies forming irregular calcified masses. Microcystic meningioma had tumor cells with thin, elongated processes encompassing microcysts and creating a cobweb-like background. Metaplastic meningioma showed cellular areas with tumor cells along with focal areas of adipose tissue. Few psammoma bodies were also seen. Chordoid meningioma had eosinophilic tumor cells present in a mucous-rich matrix. Tumor cells had vesicular nuclei, prominent nucleoli and moderate amount of cytoplasm.

**Table 22: Gender wise distribution of meningiomas [n=52]**

| Gender | Number of cases | %age |
|---|---|---|
| Male | 17 | 32.70 |
| Female | 35 | 67.30 |
| Total | 52 | 100 |

The overall incidence of meningiomas was higher in females irrespective of age and tumor type. Females constituted 35 cases [67.30%] and male to female ratio was found to be 1:2 in our study.

**Table 23: Age wise distribution of meningiomas [n=52]**

| Age [years] | Number of cases | %age |
|---|---|---|
| 21-30 | 3 | 5.76 |
| 31-40 | 4 | 7.70 |
| 41-50 | 14 | 26.92 |
| 51-60 | 16 | 30.77 |
| 61-70 | 10 | 19.23 |
| 71-80 | 5 | 9.62 |
| Total | 52 | 100 |

Age wise analysis of menigiomas showed that age group of 51-60 years was the commonest with 14 cases accounting for 30.77% of all meningiomas. The mean age was found to be 53.6 years [youngest - 26 years & oldest - 78 years].

**Table 24: Grade wise distribution of meningiomas [n=52]**

| Grade | Number of cases | %age |
|---|---|---|
| I | 44 | 84.62 |
| II | 7 | 13.46 |
| III | 1 | 1.92 |
| Total | 52 | 100 |

Grade I meningiomas were the commonest with 44 cases constituting 84.62% of all meningiomas. Female predominance was also noted in the grade I meningiomas with 35 cases [79.54%]. Transitional variant dominated the grade I category with the 29 cases [65.90%], followed by meningothelial with 7 cases [15.90%].

**Table 25: Distribution of laterality of meningiomas [n=52]**

| Side | Number of cases | %age |
|---|---|---|
| Right | 22 | 42.31 |
| Left | 30 | 57.69 |
| Total | 52 | 100 |

In our study left side was more commonly affected by the meningiomas comprising of 30 cases [57.69%] than the right side with 22 cases [42.31%].

**Table 26: Location wise distribution of meningiomas [n=52]**

| Site | Number of cases | %age |
|---|---|---|
| Frontal | 10 | 19.21 |
| Temporal | 10 | 19.21 |
| Parietal | 5 | 9.62 |
| Fronto-parietal | 4 | 7.68 |
| Parieto-occipital | 3 | 5.77 |
| Cerebellum | 3 | 5.77 |
| Olfactory groove | 3 | 5.77 |
| Posterior fossa | 3 | 5.77 |
| Occipital | 2 | 3.85 |
| Temporo-parietal | 2 | 3.85 |
| Fronto-temporal | 2 | 3.85 |
| Parasagittal | 1 | 1.93 |
| Temporal | 1 | 1.93 |
| Spheno-orbital fissure | 1 | 1.93 |
| Tuberculum sellae | 1 | 1.93 |
| Sphenoid wing | 1 | 1.93 |
| Total | 52 | 100 |

According to location wise analysis of meningiomas, both frontal and temporal lobes were common sites of involvement with 10 case each [19.21%]. Transitional meningioma [10 cases] was the commonest tumor. It was followed by parietal lobe with 5 cases. Sphenoid wing, spheno-orbital fissure & tuberculum sellae were some of the least common sites.

**Mesenchymal, non-meningothelial tumors**

One case of haemangiopericytoma showed cellular areas having tumor cells with oval to spindle shaped nuclei, conspicuous nucleoli and scant amount of slightly eosinophilic cytoplasm. These cells aggregated

around blood vessels. Mitotic count was <5/10hpf and was devoid of necrosis.
Also 2 cases of haemangioblastoma, both located in cerebellum showed an encapsulated tumor having stromal and vascular components. Stromal component was seen as large polygonal cells with vacuolated clear cytoplasm and vascular component seen as variably sized thin walled channels lined by endothelial cells. No mitosis, necrosis and pleomorphism was observed.

Another solitary case diagnosed as primitive neuroectodermal tumor [PNET] was seen in a young male of 26 years of age. Histology showed sheets of small, round, cells, hyperchromatic nuclei and scant amount of cytoplasm. Brisk mitosis, foci of necrosis and invasion into the adjacent brain parenchyma was noted. IHC revealed vimentin and CD-99 positivity. Chromogranin A and synaptophysin were negative. Ki-67 proliferation index was 70%.

**Lymphomas**

Single case of lymphomatoid granulomatosis showed expansion of blood vessel walls and dense infiltration by lymphocytes, plasma cells and eosinophils. Areas of necrosis with sheets of foamy macrophages also seen. IHC showed LCA, CD4, CD3, CD8, CD20, CD30 - positivity and CD15 was negative.

7 cases of primary CNS lymphoma [PCNSL] included in our study showed diffuse involvement of brain parenchyma by atypical lymphoid cells. Brisk mitosis and many apoptotic bodies were noted in 5 cases. 2 cases showed the infiltration and expansion of the vessel wall. The mean age for presentation was 55.5 years and male to female ratio came out to be 1:1.

6 cases of diffuse large B-cell lymphoma were included in our study and IHC was performed in 3 cases which wereLCA, CD20, MUM1,

BCL2, BCL6 - positive & CD3, CD10 negative. One case was PAX5, CD10, CD20, MUM1, CD3, EMA positive & PAN-CK, BCL6, CD138, ALK1- negative with Ki-67 proliferation index of 80% and was diagnosed as anaplastic large cell lymphoma, ALK-negative.

**Tumors of the sellar region**

Solitary case of craniopharyngioma was diagnosed and showed tumor cells arranged in broad strands and cords which were bordered by palisading columnar epithelium. Nodules of anucleate squames [wet keratin] was also identified. Surrounding brain parenchyma showed numerous Rosenthal fibers and cholesterol clefts.

**Pituitary adenoma**

22 cases of pituitary adenoma were included in our study. The mean age of presentation was 45.2 years [youngest - 22 years & oldest - 72 years]. Male to female ration came out to be 1:1.

In 7 out of 22 cases, size of the lesion was $\geq$1 cm in greatest dimension and these cases were labelled as macroadenomas. Morphology showed a cellular tumor with tumor cells arranged in sheets and pseudo-papillary pattern. Individual tumor cells had pleomorphic vesicular nuclei, prominent nucleoli and moderate amount of cytoplasm. Reticulin stain was performed in 9 cases which showed disruption of reticulin pattern in the background of tumor cells in all cases.

**Metastatic tumors**

17 cases of metastatic carcinomatous deposits were included in our study. Four cases had past history of carcinoma breast, 3 had history of carcinoma lung, 3 had history of renal cell carcinoma. Two cases

had history of carcinoma ovary and 1 case had past history of germ cell tumor. In four cases no primary was known.

The mean age of presentation came out to be 54.5 years and male to female ratio was 1:1.1. Morphology in cases of past history with breast carcinoma showed tumor cells arranged in nests and sheets, having vesicular nuclei, prominent nucleoli and moderate amount of cytoplasm. Numerous mitotic figures and areas of necrosis were noted.
In the cases with past history of carcinoma lung showed tumor cells arranged in papillae, nests and glands, showing moderate nuclear pleomorphism, vesicular chromatin, prominent nucleoli and moderate cytoplasm. No fibrillary background was noted in any section. Cases with past history of renal cell carcinoma showed tumor cells in nests separated by thin fibrovascular septae having round hyperchromatic nuclei with abundant amount of clear cytoplasm and well defined cytoplasmic boundaries.
IHC was performed on 9 cases of metastatic carcinomatous deposits. Cases diagnosed as metastatic deposits from lung were CK7, TTF-1 positive and metastatic deposits from breast showed ER, PR, HER2/neu positivity. Metastasis from ovarian origin showed PAX8, p16, WT1, B-catenin positivity and one cases showed p63, CEA, UROPLAKIN-II positivity, hence primary of urothelial origin was suggested.
So the study highlights the importance of histological examination of the intracranial space occupying lesions which ranges from infectious, inflammatory, vascular malformation to neoplastic.

# DISCUSSION

The term "intracranial space occupying lesion" [ICSOL] is used to describe a wide range of both infective and non-infectious conditions that can spread, invade, and take up space in the intracranial region.[68] Diagnosis of intra-cranial space occupying lesion is done by relevant history, clinical examination findings and appropriate investigations. Detecting intracranial lesions with high precision is now possible because of new technologies. This allows us to focus on the cause of intracranial space-occupying lesions.[69] However; histological examination with the help of IHC is still the gold standard for diagnosis.

The present study was conducted in the Department of Pathology, Dayanand Medical College & Hospital, Ludhiana for a period of 5 years from 1st July 2016 to 30th June 2021 [3.5 years retrospective and 1.5 years prospective]. In the due course of this study, out of 71,244 histopathology specimens received in our department, 266 [0.37%] were intracranial biopsies. These were histologically analyzed, special stains and IHC was performed wherever required.

In the present study of the 266 ICSOL's, 82.71% [220 cases] were neoplastic and 17.29% [46 cases] were non-neoplastic lesions. This finding is similar to the study done by Patil and Karandikar et al.[64] in which 83.87% of ICSOL's were neoplastic lesions. Similar studies done by Joshi H et al.[36] and Khonglah et al.[67] also had higher frequency of neoplastic ICSOL's comprising 64.58% and 86.13% respectively. However, in two studies conducted by Datta PK et al.[69] and Pathak and Batni et al.[68] non-neoplastic lesions predominated the ICSOL's, accounting for 59.90% and 53.33%, respectively.

In the present study, we found 17.29% [46 cases] non-neoplastic lesions of all ICSOL's. Arteriovenous malformations and epidural/subdural hematomas were the most common non-neoplastic lesions, constituting 23.91% each. The older age group of 51-60 years had the highest predilection of occurrence of non-neoplastic lesions. Mean age for non-neoplastic lesions was 46.4 years with male: female ratio of 2.5:1. Similar findings were noted by Datta PK et al.[69] in which

non neoplastic lesions were found in older age group of 51-70 years with male to female ratio of 1.27:1. Authors found intracerebral hemorrhage to be the most common non neoplastic lesion constituting 53.3% of all ICSOL's. Many studies in the literature have found varied etiologies of non-neoplastic ICSOL's. For example in a study by Pathak and Batni[68] infective lesions were the most common cause of ICSOL's comprising 53.33% in age group of 55-64 years.Also a study done by Khonglah, et al.[67] had 13.86% non-neoplastic lesions. Cystic lesions [36.36%] including dermoid cyst, arachnoid cyst, ependymal cyst, and hydatid cyst formed the majority of the non-neoplastic ICSOL's. The highest number of non-neoplastic lesions was encountered in the third decade, with a female predominance. This variation can be attributed to the different demographic factors.

In our study 82.71% [220 cases] were neoplastic lesions, out of which 57.27% were seen in males and 42.73% infemales with M:F ratio of 1.3:1 and mean age of presentation of 49.2 years. Our findings are in concordance with a study done by Deshpande NS et al.[70] in which mean age for neoplastic ICSOL's was 41.16 years with M:F ratio of 1.4:1. In a similar study done by Mohammed, et al.[71], 227 cases of ICSOL's had mean age of 42.9 years with M:F ratio of 1.2:1.

The major bulk of neoplastic ICSOL's was formed by primary brain tumors constituting 92.28% and secondary tumors being 7.72% in the present study. Similar results were seen by Khonglah et al.[67] and Deshpande NS et al.[70] in which predominance of primary brain tumors was seen constituting 97.57% and 90.10% respectively.

Astrocytic tumors were the commonest neoplastic ICSOL's constituting 36.45% of total primary brain tumors, followed by meningioma 25.60% and pituitary adenoma 10.80% in our study. Deshpande NS et al.[70] and Jayanti Mehta et al.[72] in their study of ICSOL's also found astrocytic tumors to be the commonest neoplastic lesions constituting 29.70% and 38.23% respectively. Similarly, Preeti Agarwal et al.[73] and Ghanghoria et al.[74] observed predominance of astrocytic tumors constituting 29.6% and 24.61% respectively. However, discordance was noted with the studies done by Mohammed, et al.[71] and

Kakshapati T et al.[75] which showed higher incidence of meningioma, 30.8% and 30.3% respectively.

Glioblastoma [grade IV] was the commonest astrocytic tumor in our study constituting 81.08% of all astrocytic tumors. Grade IV tumors were followed by diffuse astrocytoma [grade II] and anaplastic astrocytoma [grade III] constituting 12.16% and 6.76% respectively in our study.This finding is in concordance with the studies done by Deshpande NS et al.[70] and Adnan H.A. et al.[32] where the predominance of glioblastoma among all astrocytic tumors was noted constituting 63.33% and 61.53% respectively. Also in a similar study by Hema Neelakantaiah Anadure et al.[43] had 29.16% cases of grade IV GBM, followed by diffuse astrocytoma and anaplastic astrocytoma with 12.50% and 8.33% cases respectively. Vimal et al.[76] found predominance of diffuse astrocytoma followed by GBM having 18.8% and 13.3% respectively.

Mean age for astrocytic tumors was found to be 43.6 years and 45.2 years respectively in studies done byKakshapati T et al.[75] and Mohammed et al.[71]. This is comparable to our results as the mean age for the astrocytic tumors was found to be 52.9 years. The M:F ratio showed male predominance in both the above mentioned studies [1.4:1 in Kakshapati T et al.[75] and 4:1 in Mohammed, et al.[71]] . Our findings are concordant to these studies with M:F ratio of 2.5:1.

In present study the commonest site of occurrence of astrocytic tumors was frontal lobe followed by temporal lobe comprising 35.10% and 23% respectively. This is similar to the observations made by Larjavaara S et al.[77] in which the astrocytic tumors showed frontal lobe [40%] as most frequent site of involvement followed by temporal lobe [29%]. In a study done by Peter D. Inskipet al.[78] most of the astrocytic [62%] and oligodendroglial [11%] tumors were seen on the left side which was also observed in the present study.

IDH mutation testing is rapidly becoming a mainstay of the pathological workup of human brain tumors. It can be used to diagnose a tumor as well as predict how well it will respond to treatment.[79] In WHO grade II and III gliomas and secondary glioblastomas, IDH mutations were

common, while they were less common in primary glioblastomas. In our study, one case of diffuse astrocytoma and one case of GBM were IDH mutant, while the rest of the cases were IDH wildtype.

Oligodendroglial tumors comprised 9.87% of all primary tumors in our study andoligodendroglioma was the commonest tumor constituting 75% of all oligodendroglial tumors followed by15% cases of anaplastic oligodendroglioma and 10% cases of anaplastic oligoastrocytoma. Comparable results were seen in a study by Chen et al.[80] who retrospectively studied 34140 cases of brain tumors and found 56.25% cases of oligodendroglioma, 30.23% cases of anaplastic oligodendroglioma and 5.03% cases of anaplastic oligoastrocytoma with additional 13.75% cases of oligoastrocytoma. Pai T et al.[81] found 26-40 years to be the commonest age group for oligodendroglioma which is comparable to 31-40 years in the present study. This study also had male predominance for olgodendrglial tumors with male to female ratio of 1.8:1 which is in concordance with M:F ratio of 2.3:1 of the present study.

Frontal lobe was found to be the most common site of occurrence of olgodendrglial tumors in the current study, accounting for 60% of cases, while Pai T et al.[81] found 46.8% of the cases in frontal lobe. In our study, grade II oligodendroglialtumors outnumbered grade III tumors constituting 75%, and similar findings are reported by Chen et al.[80], who found 66.43% of grade II and 33.57% of grade III oligodendroglial tumors. Pai T et al.[81], on the other hand, found grade III oligodendroglial tumors predominance, which contradicted our findings.

One of the strongest indicators of oligodendroglial tumors is the 1p/19q co-deletion. Radio-chemotherapy with procarbazine and vincristine for anaplastic oligodendroglial tumours uses 1p/19q as a biomarker.[82] This information was unavailable to us due to a lack of molecular genetics tools in our facility.

Our study also included 1.97% cases of pilocytic astrocytoma, out of which 3 showed younger age of presentation i:e 5, 11 and 14 years and 1 case was seen in 40 years of age. The mean age came out to be

17.5 years with M:F ratio of 1:1. Our results are in concordance with the study done by Chen et al.[80] which showed 1.37% cases of pilocytic astrocytoma with mean age of 17 years. However their study showed slight male predominance with M:F ratio of 1.1:1.

Out of 82.71% cases of neoplastic ICSOL's, 1.81% were ependymal tumors in the present study. These tumors generally have a low cell density and a low mitotic count and very rarely invade adjacent CNS parenchyma.[44] Mean age of presentation was 26.5 years with male to female ratio of 3:1. Our findings arein concordance with the study done by Hamdani et al.[63] who observed 3.4% cases of ependymal tumors with M:F ratio of 3:1.

A solitary case of ganglioglioma in a 48 year old male was included in our study. According to the available data ganglioglioma is a rare entity and it accounts for only 0.4% of all brain tumors.[27] Morphology showed a cellular tumor with tumor cells in diffuse sheets against a fibrillary background intermingled with dysplastic neurons. Studies done by Pidakala P. et al.[83], Kakshapati T et al.[75] and Poonia et al.[84] also observed a solitary case of ganglioglioma in their study.

Medulloblastoma is a neuroepithelial embryonal tumor that develops in the cerebellum or dorsal brain stem. They can occur at any age, but are more common in children. The current study had 1.48% cases of medulloblastoma, with a male predominance [M:F ratio of 2:1] and mean age of 17 years. Chen et al.[80] had 1.85% cases of medulloblastoma, with a mean age of 15.4 years and an M:F ratio of 1.7:1. These findings are almost similar to the present study.

Schwannomas comprised 3.94% of all neoplastic lesions in the present study. Majority of them [62.50%] were located in the cerebellopontine angle and showed male predominance with M:F ratio of 3:1. Mean age came out to be 44 years. The results are concordant with the study done by Renu Thambi et al.[62] which had 13.9% cases of schwannomas and the most common site was cerebellopontine angle with median age of 48 years. However, their M:F ratio was 1:1. Similarly Chen et al.[80] and Khonglah, et al.[67] also found that mean age of presentation of

schwannoma to be 45 years and 44 years respectively in agreement with our findings.

Meningiomas are usually benign, slowly growing localized tumors, however they can cause significant morbidity and, in rare cases, mortality. Even benign meningiomas have morbidity and mortality risks in addition to gaining anaplastic characteristics. In the present study, transitional meningiomas formed the major bulk and comprised 55.77% of all meningiomas. Studies done by Poonia et al.[84] and Nitin Maheshvar Gadgil et al.[85] showed similar results with transitional meningiomas being the most common, comprising 32.25% and 24.2% respectively, followed by meningothelial meningiomas constituting 19.35% and 22.6% respectively. Present study showed 13.46% meningothelial meningiomas.

Females outnumbered the males in all the grades of meningiomas in the present study with male to female ratio to be 1:2. These results are very similar to the study done by Niranjan J et al.[86] in which M:F ratio was 1:1.7. Meningiomas were found to be more common in 4$^{th}$ and 5$^{th}$ decade of life in our study, accounting for 57.69% cases combined. Our findings are in absolute concordance with the observations by Niranjan J et al.[86] and Narmadha R at al.[87]. These studies also showed predominance of meningiomas in 4$^{th}$ and 5$^{th}$ decade of life.

Grade I meningiomas [84.62%] were the commonest and transitional variant dominated the grade I category with the 65.90% cases, followed by meningothelial variant with 15.90% cases in our study. Study done by Niranjan J et al.[86] showed concordance with our results with 92.98% cases of grade I meningioma. However, meningiothelial variant was the most common subtype with 33.33% cases in their study.

In the present study left side was more commonly affected by the meningiomas [57.69% cases] than the right side [42.31% cases], along with frontal and temporal lobe being the most common site with 19.21% cases each. However, Peter D. Inskip et al.[78] in the study on brain tumor laterality showed, 52.07% of meningiomas occurring in left side of the brain.

Walker and Davis[88] in 2019 had 4.82% [585 cases] of CNS lymphomas, affecting 57.26% males and 42.74% females with M:F ratio of 1.3:1. In the present study lymphomas constituted 3.94% of all neoplastic ICSOL's, with M:F ratio of 1:1. Our finding is almost similar to the above study.

Pituitary adenomas constitute 10%–15% of all intracranial neoplasms in most neurosurgery practices. The vast majority of adenomas are sporadic.[44,49] In our study these tumors constituted 10.80% [22 cases] of all neoplastic ICSOL's. 31.81% of these lesions were coined as pituitary macroadenoma due to their large size. The mean age of presentation was 45.2 years [youngest - 22 years & oldest - 72 years]. Male to female ration came out to be 1:1. Kakshapati T et al.[75] had 13.6% cases of pituitary adenoma in their study and M:F ratio was 1:1 and 41-60 years age bracket was the most common for presentation. Similarly in another study done by Renu Thambi et al.[62] mean age of presentation was 47 years. So findings of present study are concordant with other studies in the literature.

There were 7.72% cases of metastatic brain tumors in the present study and most common histological type was adenocarcinoma [88.23% cases]. Metastatic tumors were common after fifth decade of life. No cases of metastatic deposits were reported in less than 30 years of age. The most common primaries were from breast and lungs followed by renal in origin. Mean age of metastatic brain tumors in our study came out to be 54.5 years with slight female dominance [M:F 1:1.1]. In a similar study done by Mohammed, et al.[71] in 2021, 7.7% cases of ICSOL's were metastatic tumors. Most common histological type was found to be adenocarcinoma [82.3%] and mean age was 47.3 years. While Vimal et al.[76] found most cases of squamous cell carcinoma [71.42%] with origin from lung as metastatic tumor of the brain.

Another study done by Kakshapati T et al.[75] had 5.88% metastatic tumors and M:F ratio was found to be 1.1:1. Chen et al.[80] showed male predominance in metastatic tumors with M:F ratio of 1.6:1 and mean age was 55 years which is in concordance with the present study.

In the present study radiological concordance with histological diagnosis was 79.70%. Sunila et al.[64] and Dogar t et al.[57] observed a concordance of 82.95% and 68% with the histopathological diagnoses.

So the present study concludes that although there is wide variety of non-neoplastic and neoplastic intracranial lesions, the definitive diagnosis can only be made by histopathological examination of the tissue aided with special stains and IHC.

# SUMMARY AND CONCLUSION

Intracranial space occupying lesions [ICSOL] are the leading cause of neurological morbidity and mortality. These can be neoplastic and non-neoplastic. In this study we analyzed 266 cases of both non-neoplastic and neoplastic ICSOL's and the findings are-

1. A total of 71,244 specimens were received in the department, out of which 266 were intracranial lesions.
2. Out of the 266 ICSOL's, 82.71% [220 cases] were neoplastic and 17.29% [46 cases] were non-neoplastic lesionswith mean age of 48.5 years.
3. Arteriovenous malformation and epidural/subdural hematomawere the most commonly occurring non-neoplastic ICSOL's in our studyconstituting 23.91% each.
4. Age group of 51-60 years had the highest predilection of occurrence of non-neoplastic lesions with mean age of 46.4 years and M:F ratio of 2.5:1.
5. Temporal lobe was found to be the commonest site for non-neoplastic lesions with AVM being the most common lesion.
6. The neoplastic ICSOL's were divided into primary and secondary tumorswith 203 cases [92.28%] and 17 cases [7.72%] respectively and GBM [grade IV] being the most common primary astrocytic tumor with 60 cases [29.6%].
7. Astrocytic tumors showed highest tendency of occurrence in age group of 51-60 years followed by 61-70 years. The least affected population was the patients of the eighth and ninth decade.
8. Frontal lobe was the commonest site for astrocytic tumors [35.17%] followed by temporal lobe [23%].
9. Oligodendroglioma was the commonest oligodendroglial tumor constituting 7.40% of all primary tumors and showed M:F ratio of 2.3:1.
10. Pilocytic astrocytoma showed predilection for young population with commonest site being the posterior cranial fossa.

11. Ependymal tumors had 2 cases each of ependymoma and anaplastic ependymoma.
12. A solitary case of ganglioglioma in a 48 year old male was observed.
13. Cerebellopontine angle was found to be the commonest site for schwannomas comprising 62.50% cases and a mean age of 44 years. Antoni-A and Antoni-B areas were present in all cases and 3 out of 8 cases also showed Verocay bodies.
14. Transitional subtype formed the major bulk in meningiomas with 55.77% followed by meningothelial and angiomatous variants.
15. The overall incidence of meningiomas was higher in females irrespective of age and tumor type and male to female ratio was found to be 1:2.
16. Grade I meningiomas were the commonest constituting 84.62% of all meningiomas.
17. Primary CNS lymphoma [PCNSL] comprised of 7 cases with mean age for presentation of 55.5 years and male to female ratio of 1:1.
18. There were 22 cases of pituitary adenoma, 7 had size of the lesion >1 cm in greatest dimension and these cases were labelled as pituitary macroadenomas.
19. Metastatic brain tumors constituted 7.72% cases and most common histological type was adenocarcinoma.
20. Metastatic tumors were common after fifth decade oflife. No cases of metastatic deposits were reported in less than 30 years of age. The most common primaries were from breast and lungs followed by renal in origin.
21. Radiological correlation with histopathological diagnosis was 79.70%.

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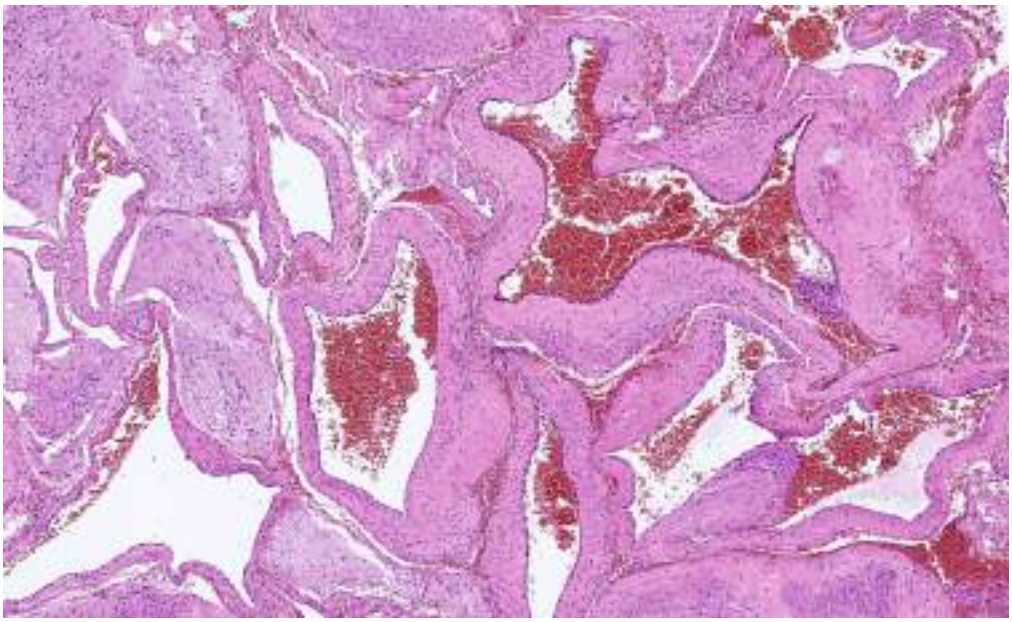
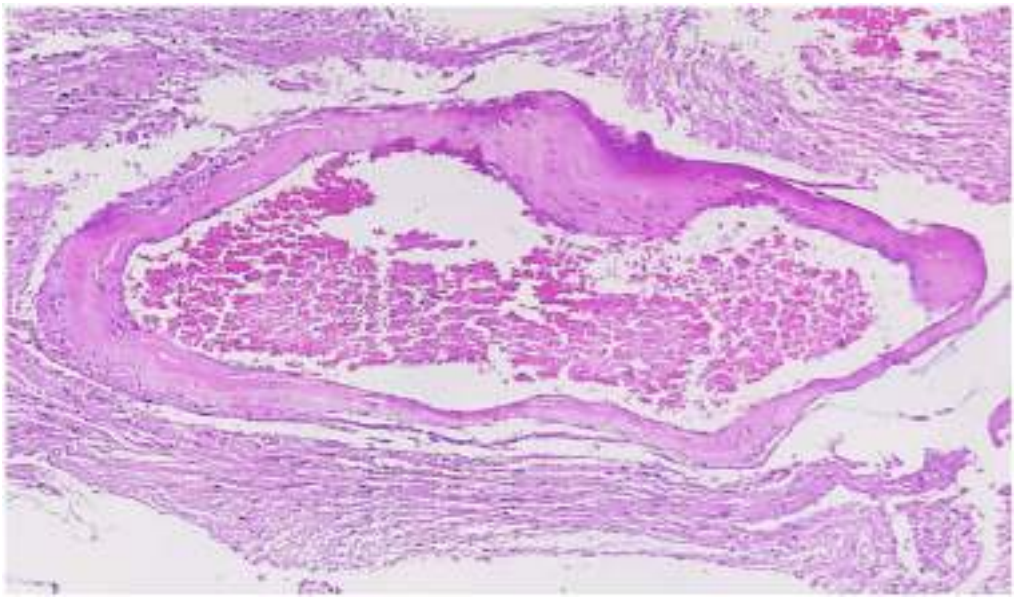
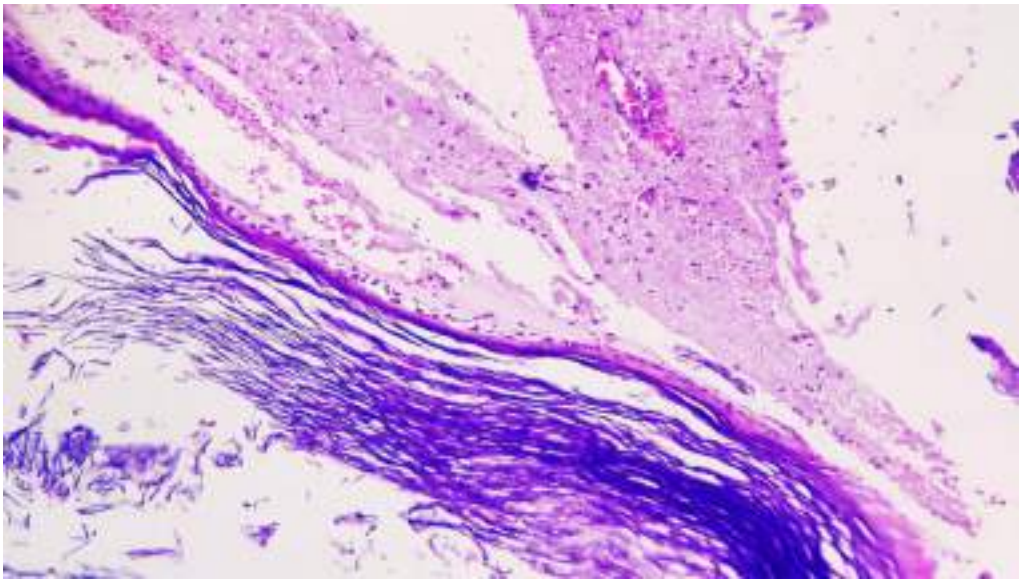


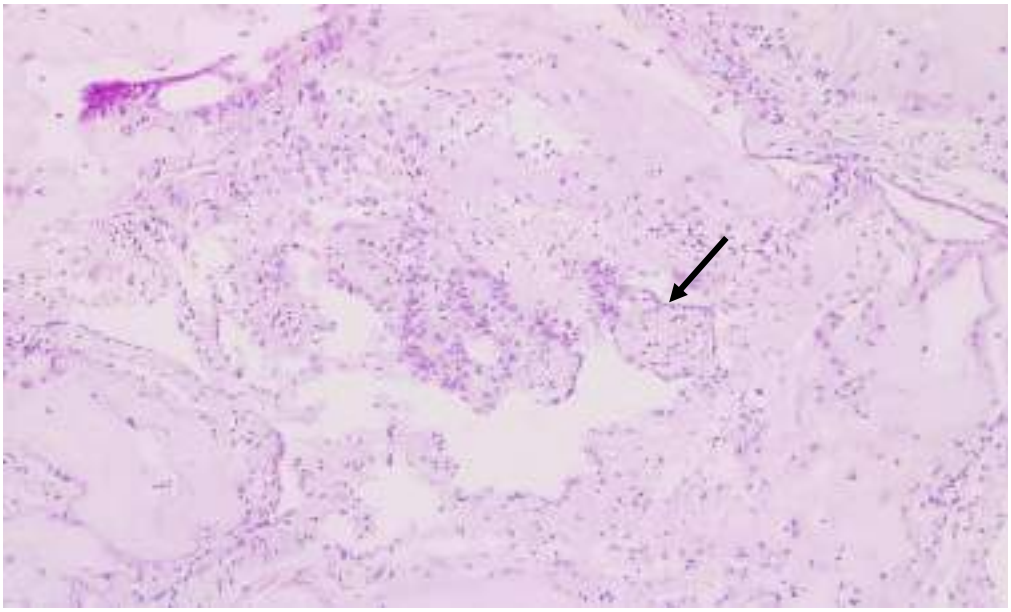
**Picture 1: Arteriovenous malformation – Micrograph showing numerous dilated and congested blood vessels having variable wall thickness. [H&E 100X]**



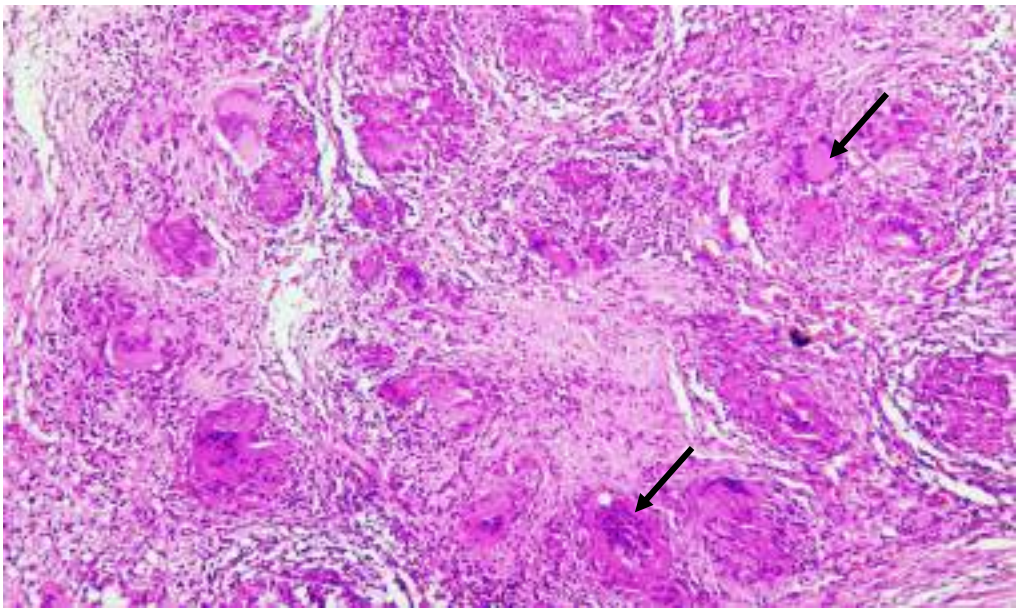
**Picture 2: Arteriovenous malformation – Micrograph showing dilated and congested blood vessel having variable wall thickness. [H&E 400X]**



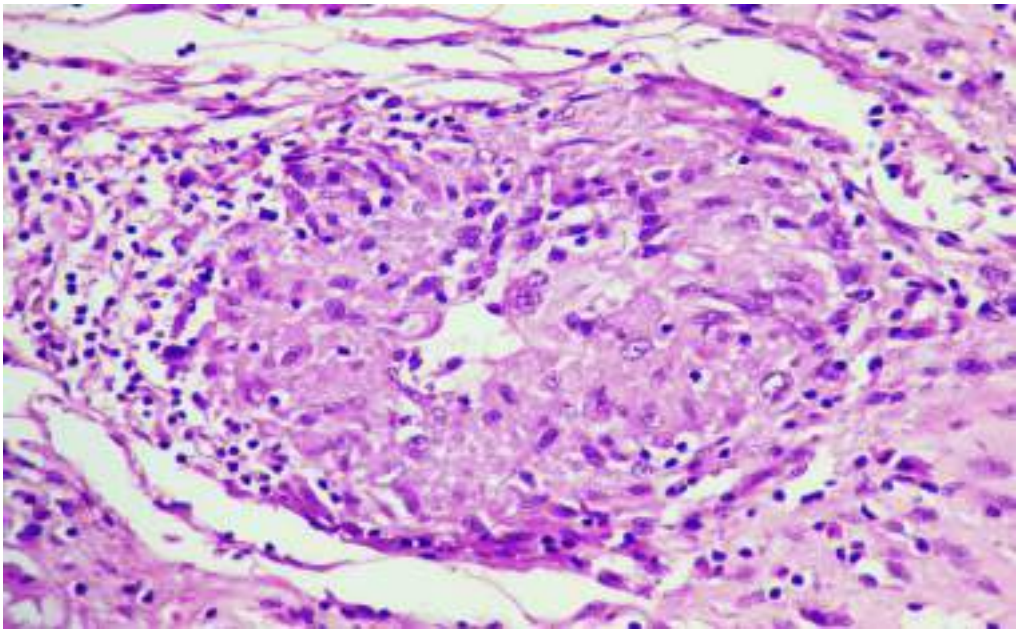
**Picture 3: Epidermal inclusion cyst–Micrograph showing acyst wall lined by keratinized squamous epithelium. [H&E 100X, 400X]**



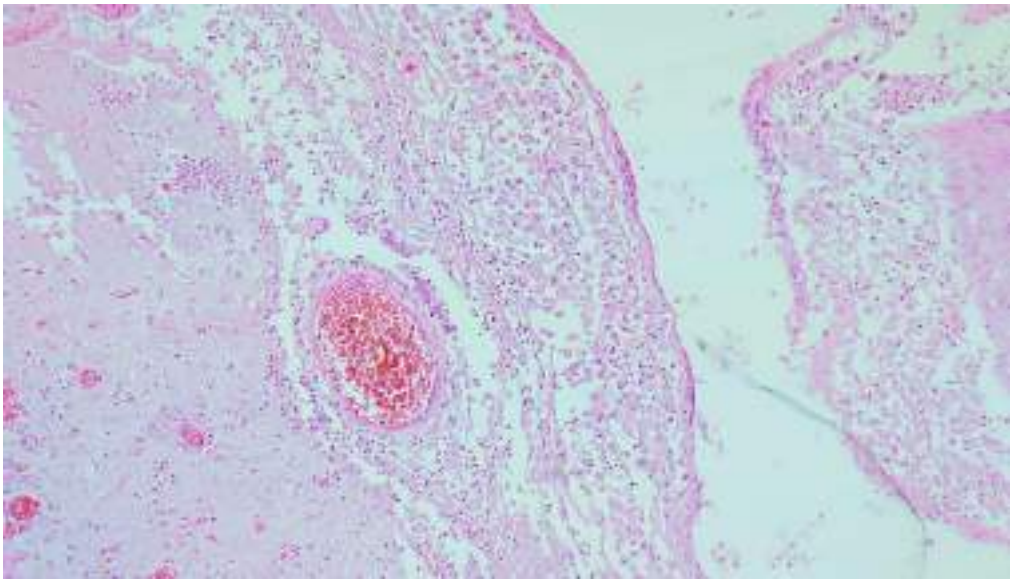
**Picture 4: Arachnoid cyst – Micrograph showing a cyst wall lined by flattened epithelium with focal meningothelial cells [arrow]. [H&E 100X]**



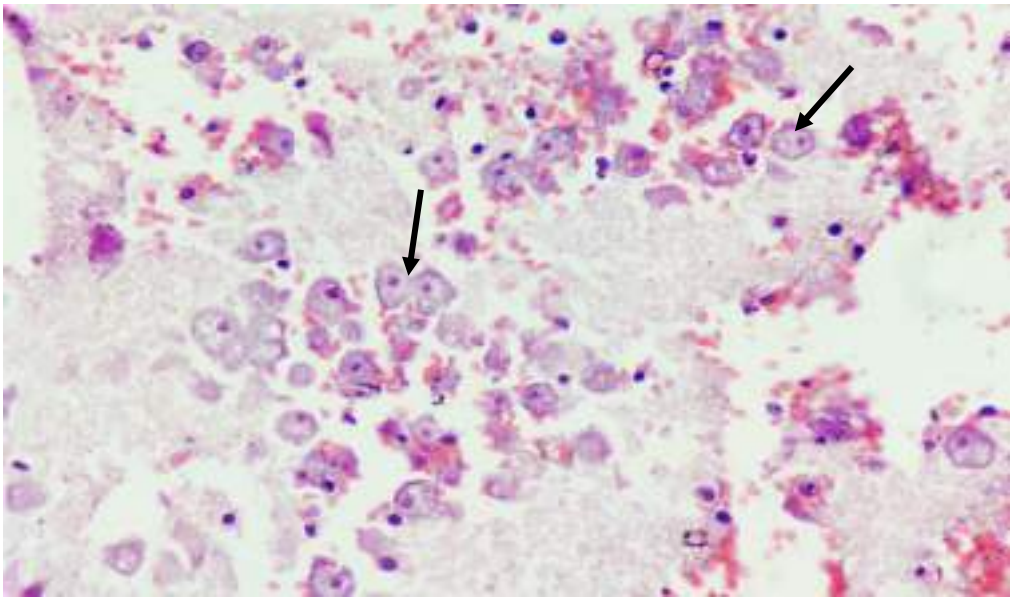
**Picture 5: Tuberculosis - Brain parenchyma showing numerous collections of epithelioid cells forming granulomas with foreign body and Langhan's type giant cell reaction [arrow]. [H&E 100X]**



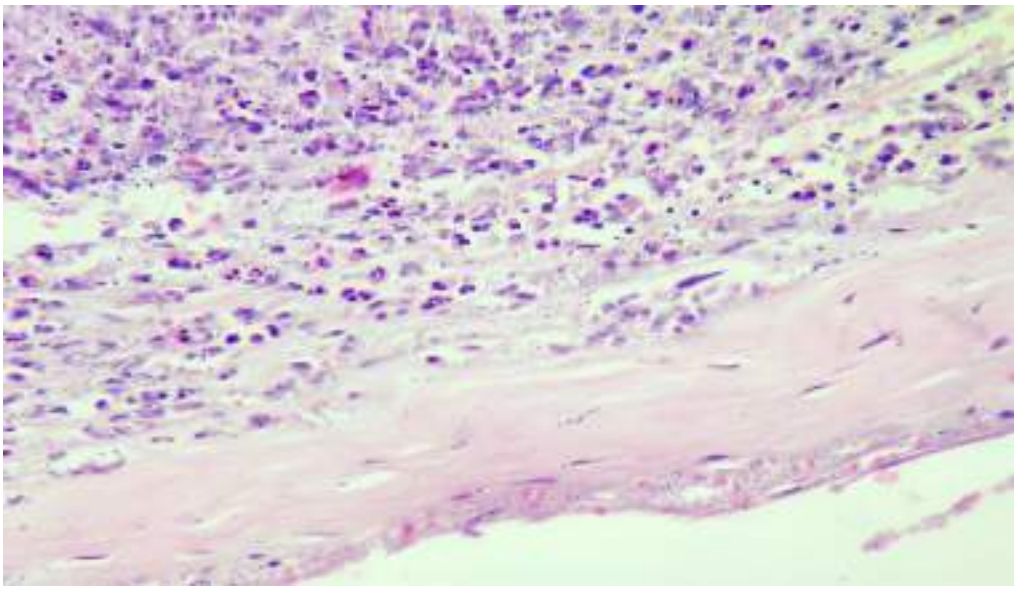
**Picture 6: Tuberculosis - Epithelioid cell granuloma surrounded by lymphocytic collar. [H&E 400X]**



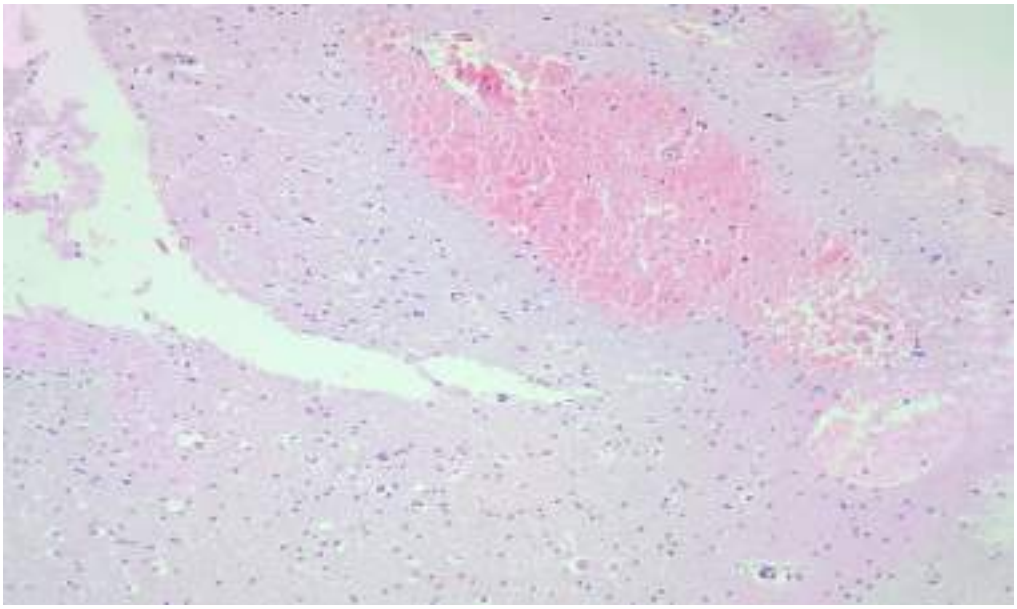
**Picture 7: Amoebic abscess- Brain parenchyma showing numerous trophozoites of Entamoeba histolytica with dense inflammatory granulation, acute and chronic. [H&E 100X]**



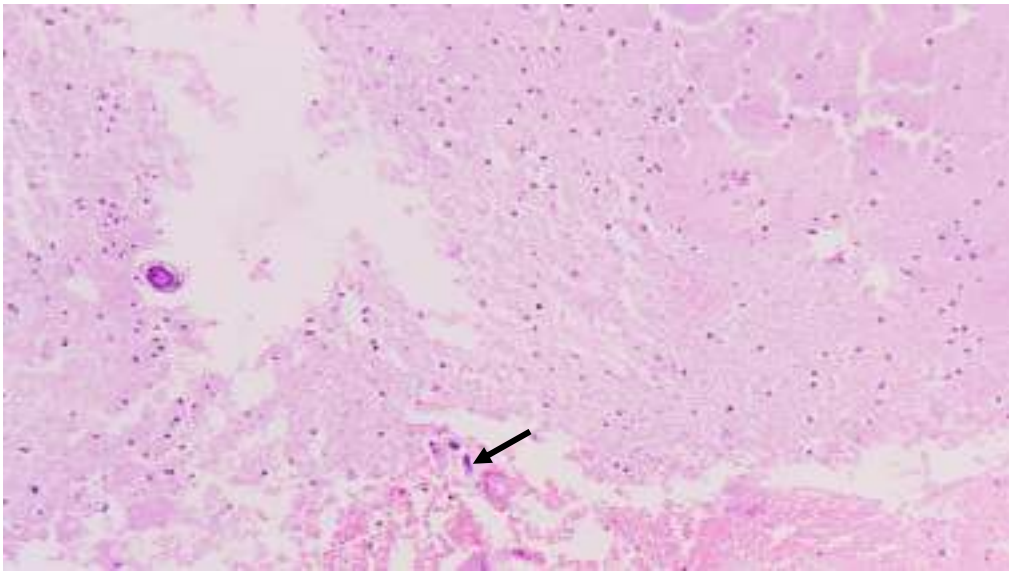
**Picture 8: Amoebic abscess- Trophozoites of Entamoeba histolytica [arrow]. [PAS 400X]**



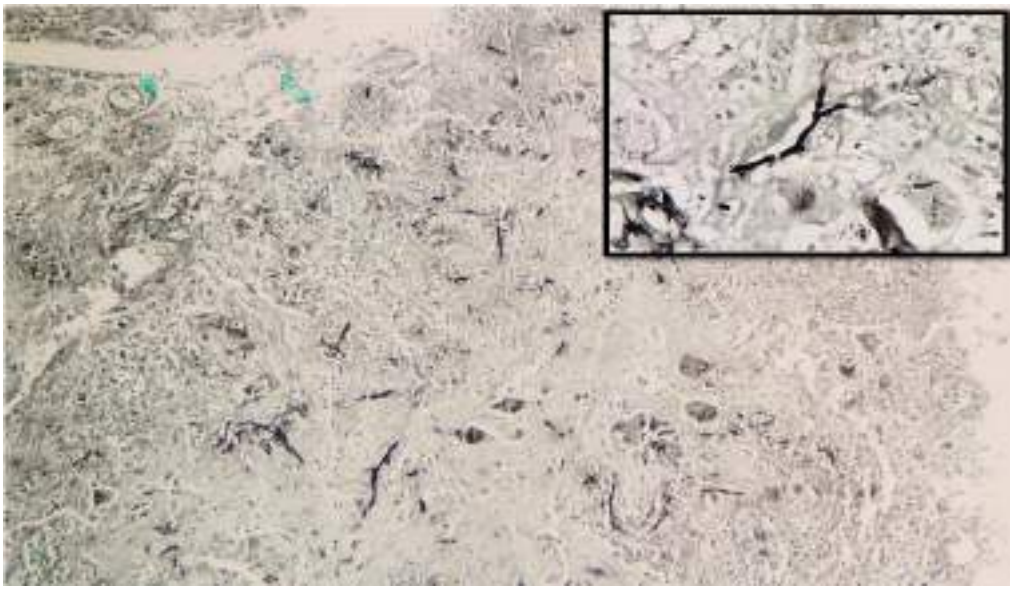
**Picture 9: Pachymeningitis – Dura mater showing infiltration by inflammatory cells mainly neutrophils. [H&E 100X]**



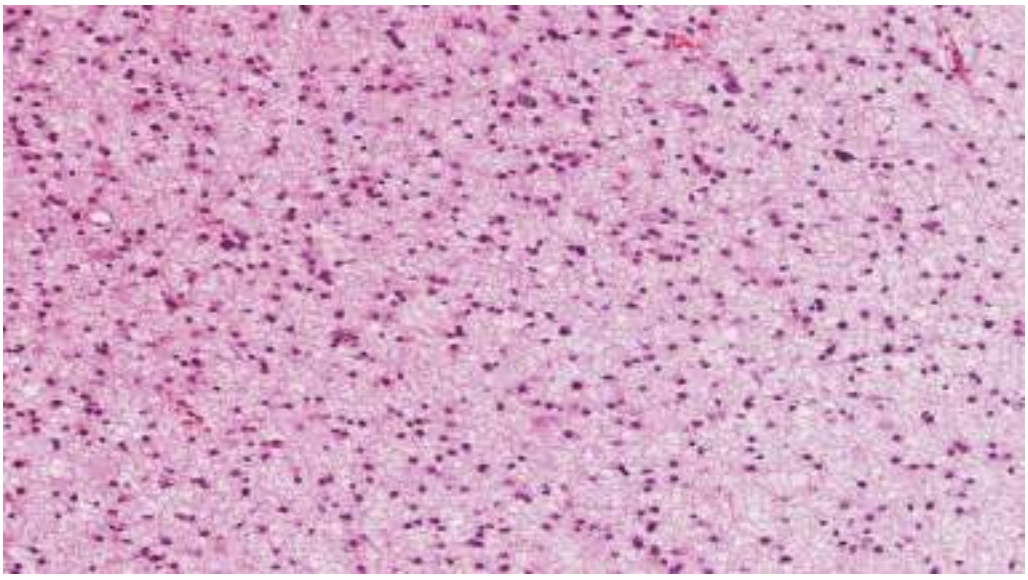
**Picture 10: Intrparenchymal hemorrhage - Brain parenchyma showing intraparenchymal hemorrhage with focal areas of necrosis [H&E 100X]**



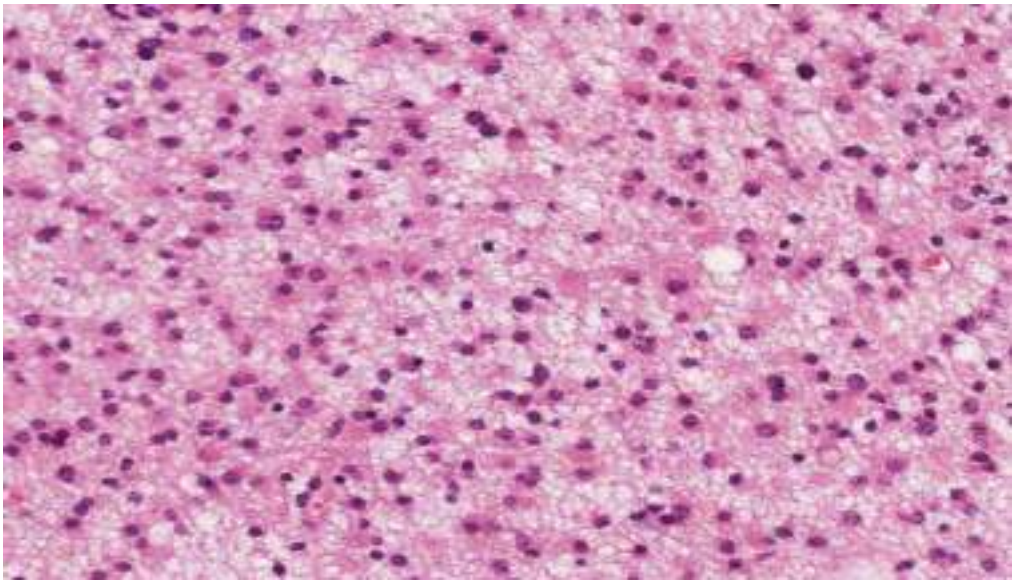
**Picture 11: Aspergillosis- Brain parenchyma showing septate fungal hyphae [arrow]. [H&E 100X]**



**Picture 12: Aspergillosis–Brain parenchyma showing many septate, acute angle branching fungal hyphae [arrow]. [GMS 100X, 400X]**

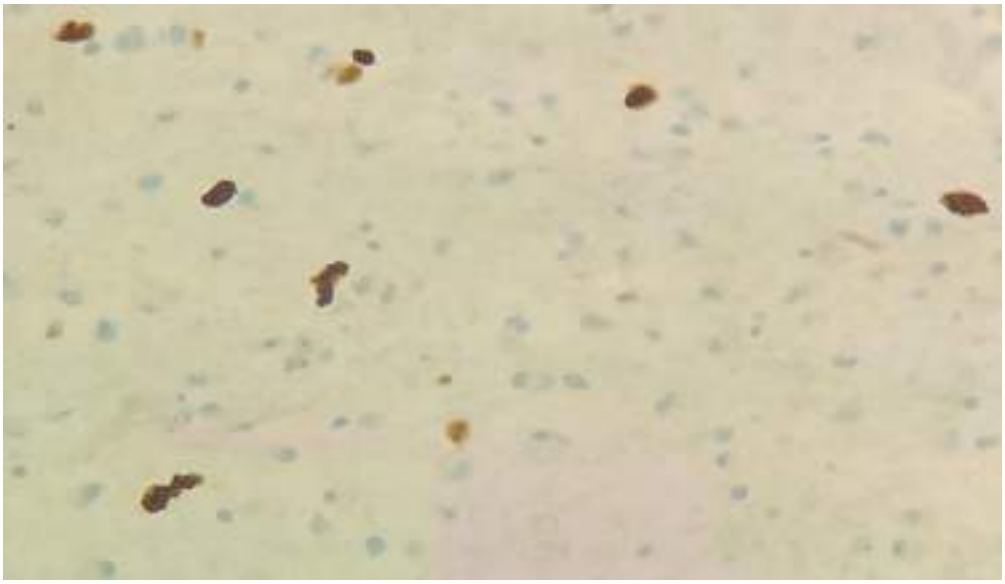


**Picture 13: Diffuse Astrocytoma- Cellular tumor with cells arranged in sheets against a fibrillary background. [H&E 100X]**

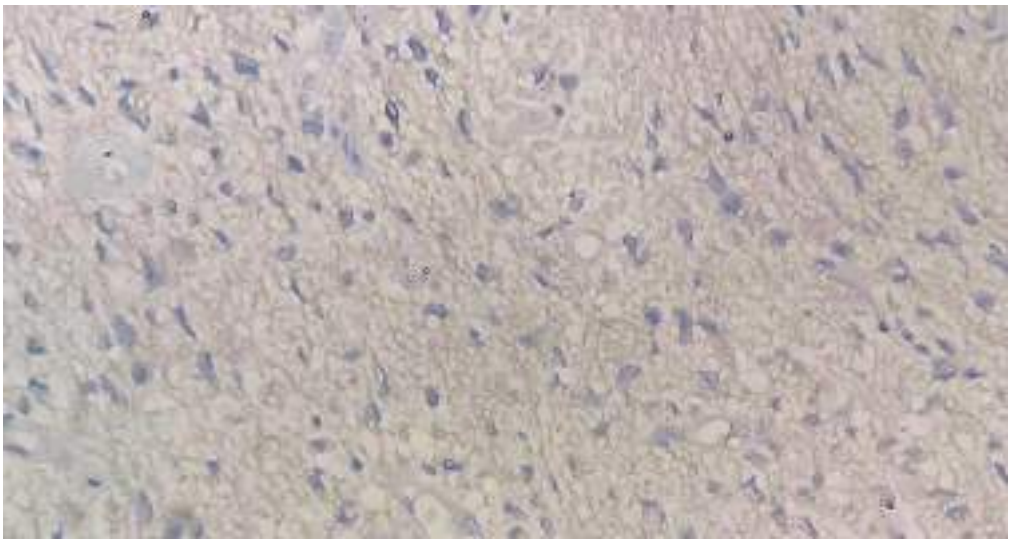


**Picture 14: Diffuse Astrocytoma- Tumor cells exhibiting moderate nuclear pleomorphism and hyperchromasia. [H&E 400X]**

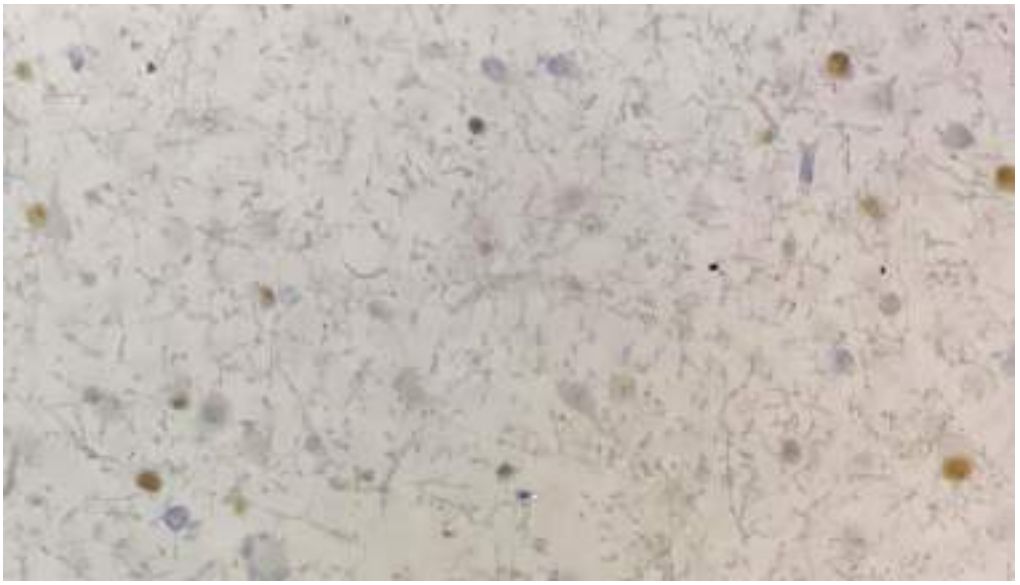
**Picture 15: Diffuse Astrocytoma- Tumor cells showing strong cytoplasmic GFAP positivity. [100X]**



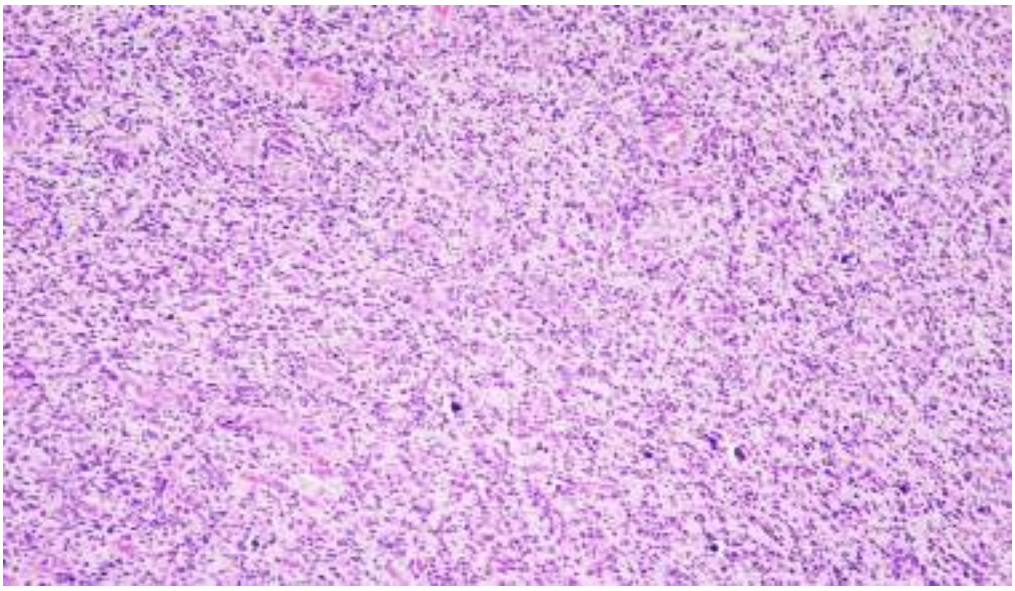
**Picture 16: Diffuse Astrocytoma- Ki-67 proliferation index 3%. [400X]**



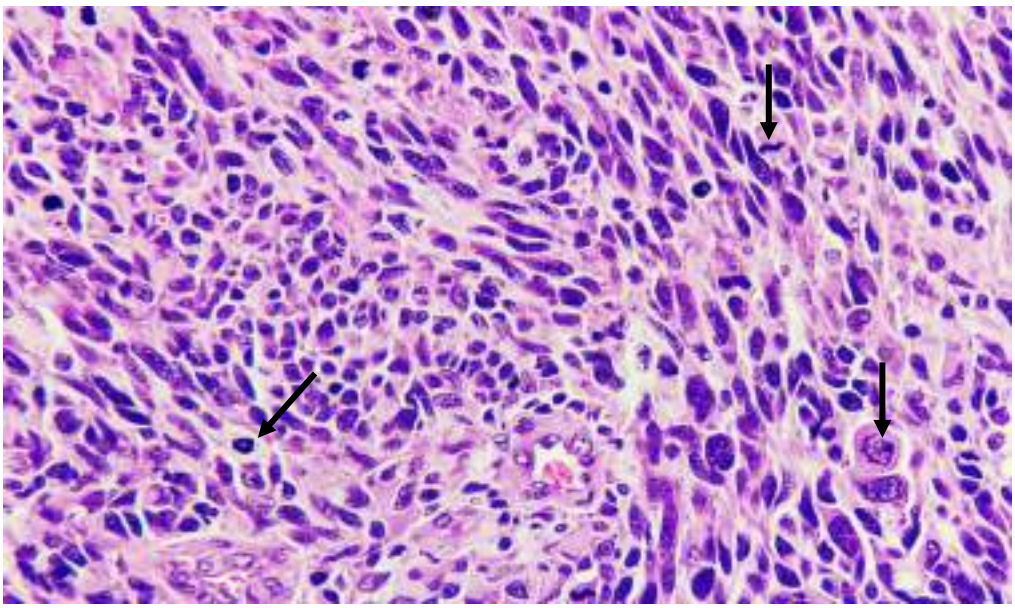
**Picture 17: Diffuse Astrocytoma- Tumor cells showing IDH-1 negativity. [400X]**



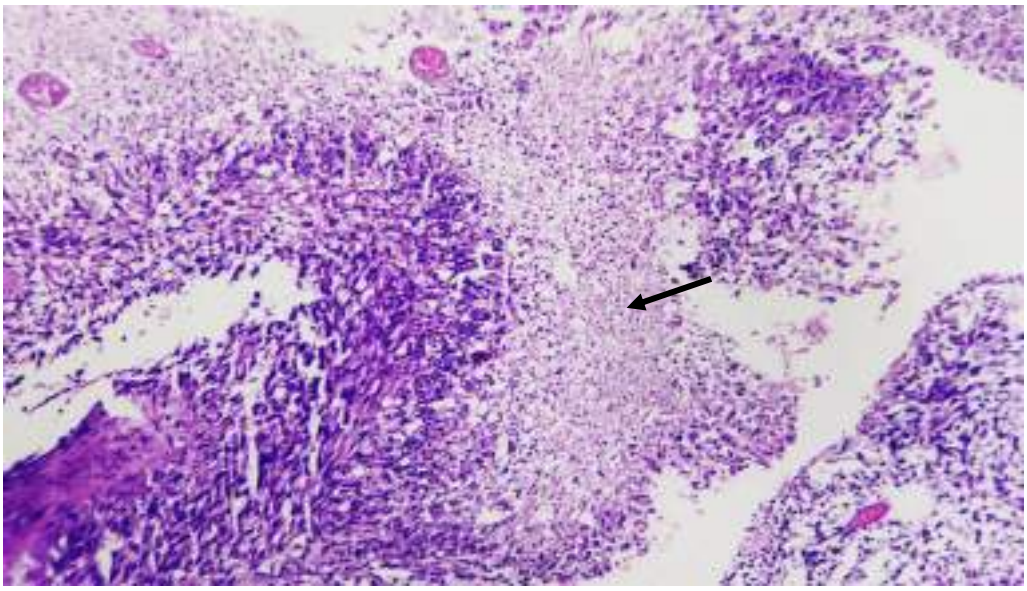
**Picture 18: Diffuse Astrocytoma- Tumor cells showing ATRX negativity. [400X]**



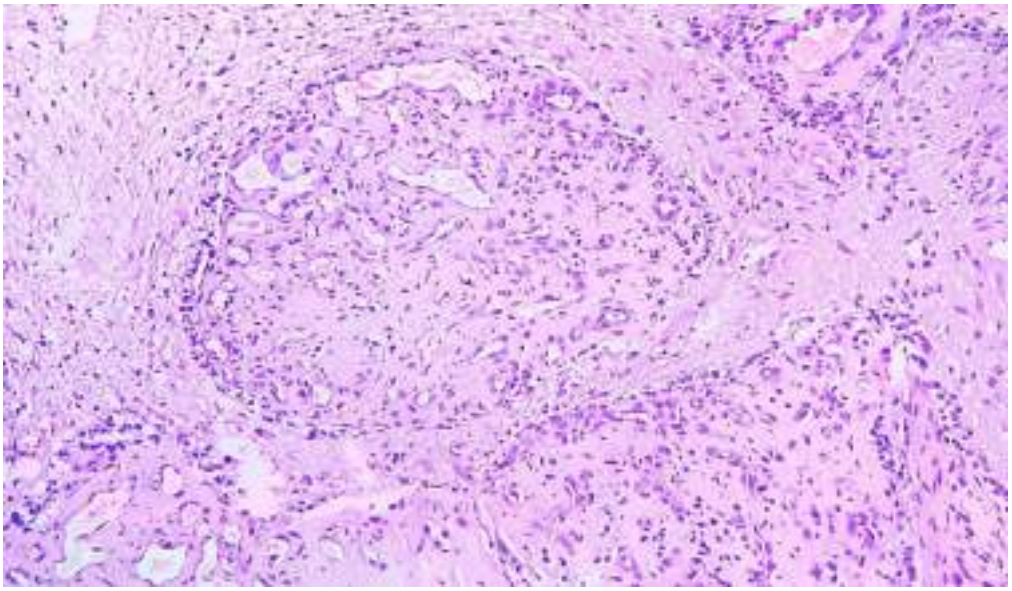
**Picture 19: Glioblastoma – Highly cellular tumor, cells arranged in sheets. [H&E 100X]**



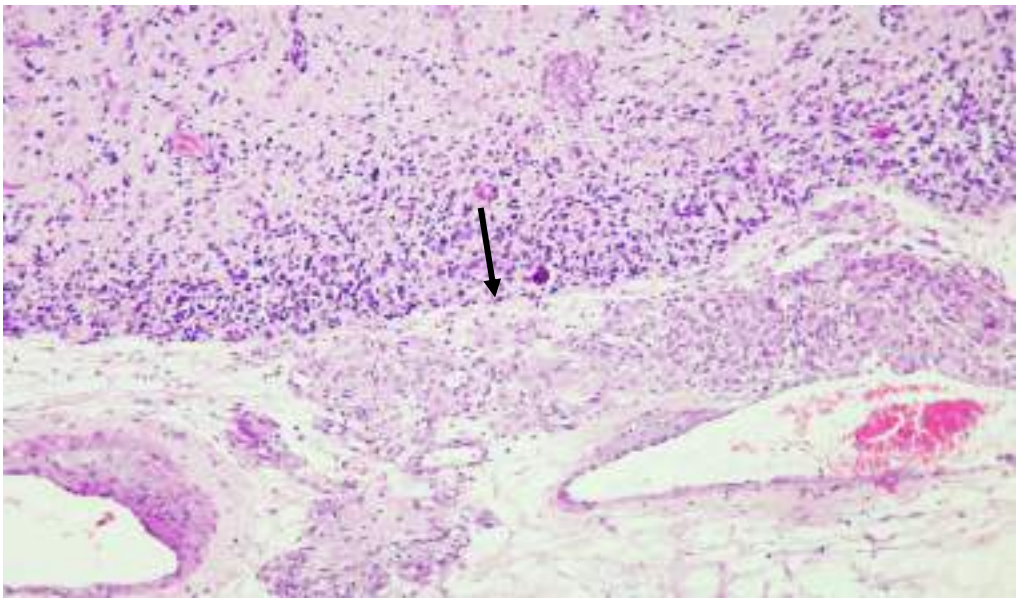
**Picture 20: Glioblastoma –Cellular tumor areas showing nuclear pleomorphism, hyperchromasia with brisk mitosis [arrow]. [H&E 400X]**



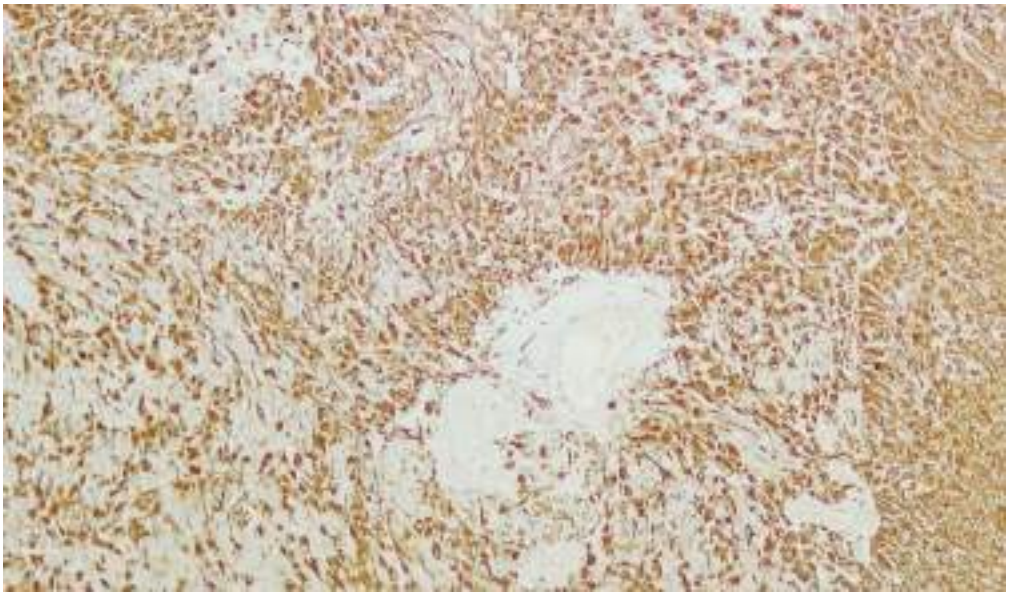
**Picture 21: Glioblastoma –Tumor showing geographic necrosis [arrow] palisaded by tumor cells. [H&E 100X]**



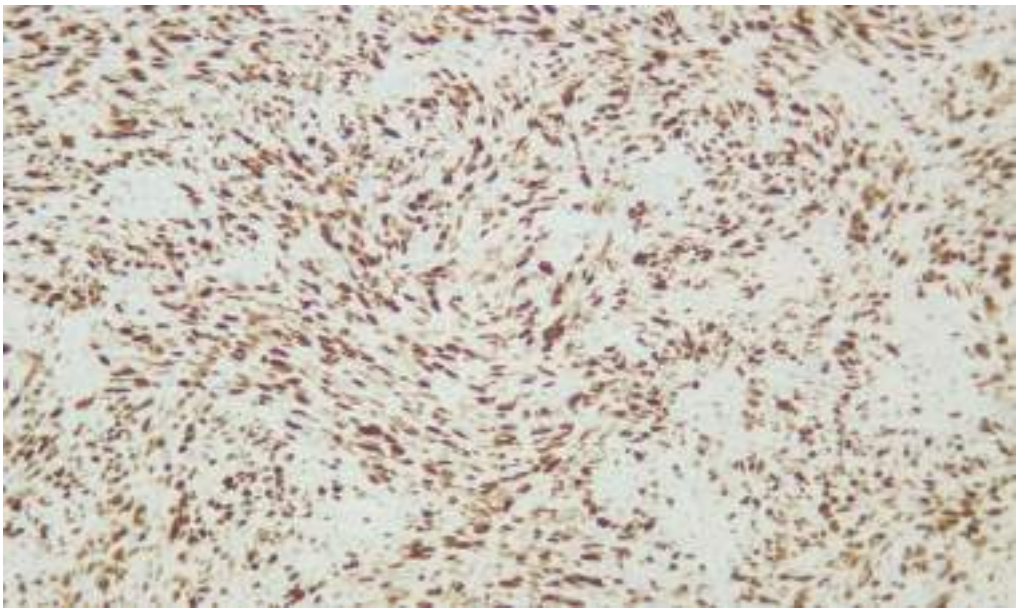
**Picture 22: Glioblastoma– Tumor showing prominent microvascular proliferation. [H&E 400X]**



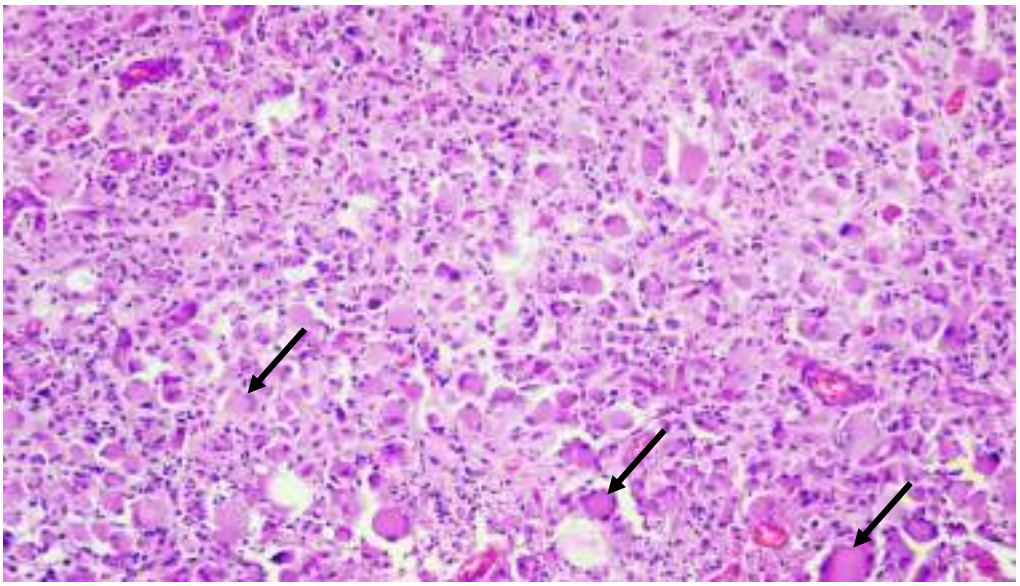
**Picture 23: Glioblastoma– Tumor infiltrating the adjacent brain parenchyma and reaching sub-pial location [arrow]. [H&E 100X]**



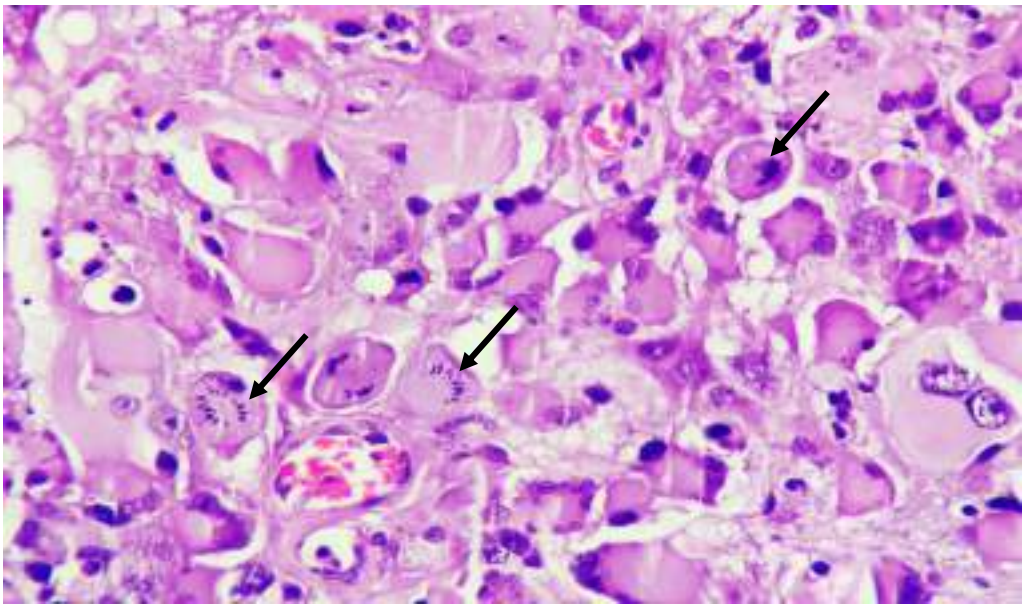
**Picture 24: Glioblastoma– Tumor cells showing strong cytoplasmic GFAP positivity. [100X]**



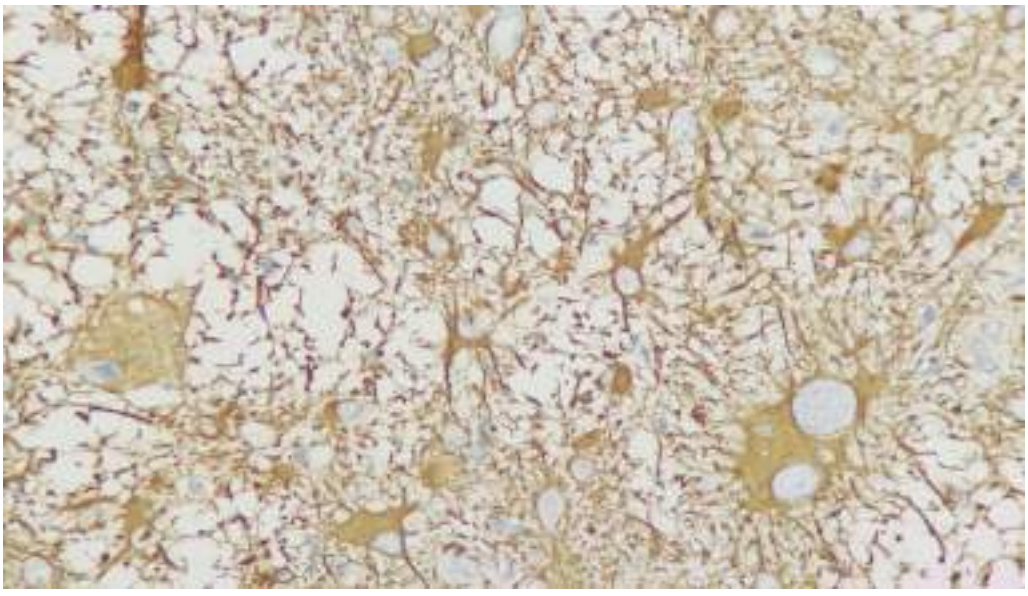
**Picture 25: Glioblastoma - Ki-67 proliferation index 80%. [100X]**



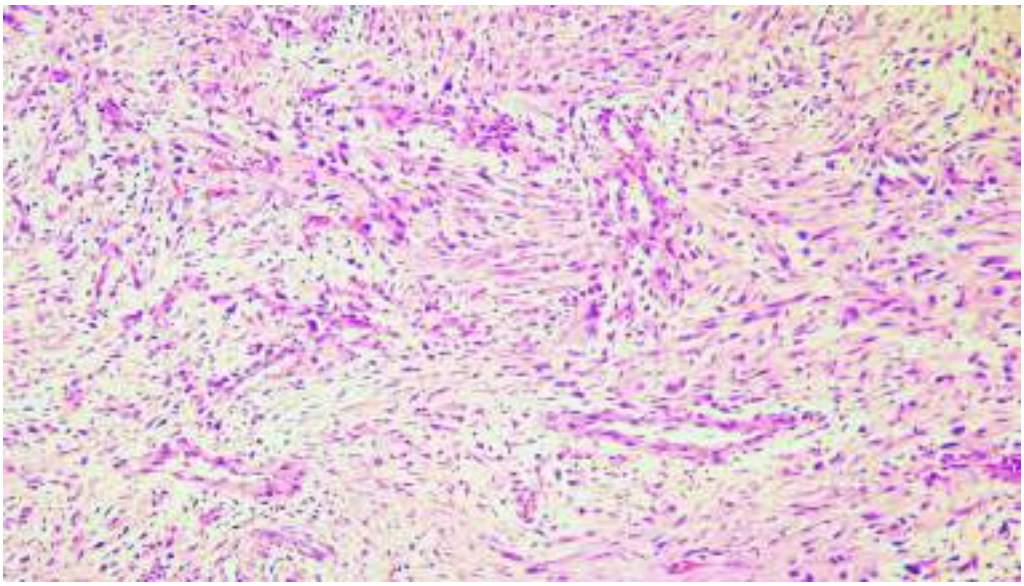
**Picture 26: Giant cell Glioblastoma – Cellular tumor with numerous multinucleated giant cells [arrow][H&E 100X]**



**Picture 27: Giant cell Glioblastoma – Brisk mitotic activity [arrow] and multinucleated giant cells [H&E 400X]**

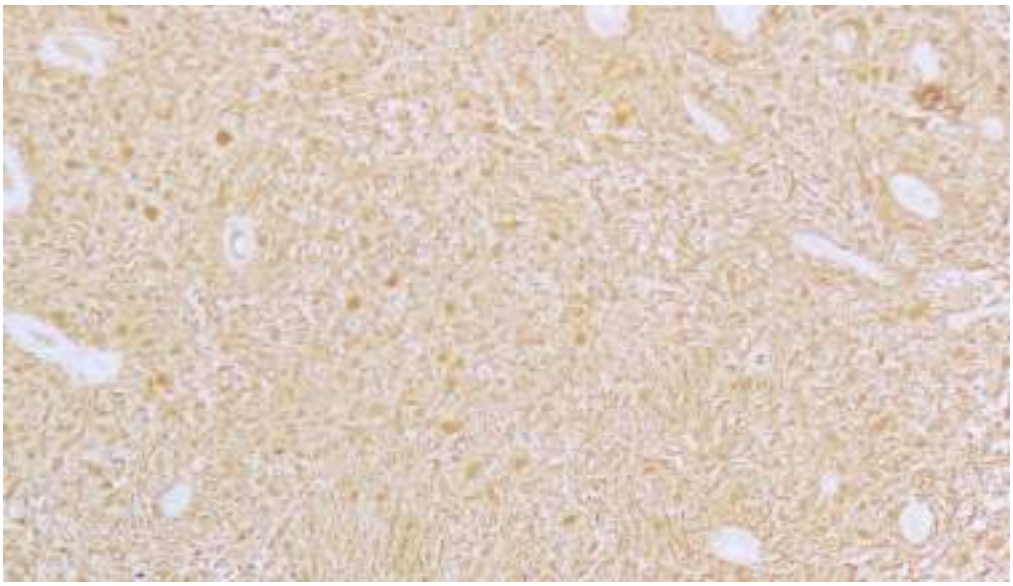


**Picture 28: Giant cell Glioblastoma – Diffuse strong cytoplasmic GFAP immunoreactivity. [100X]**

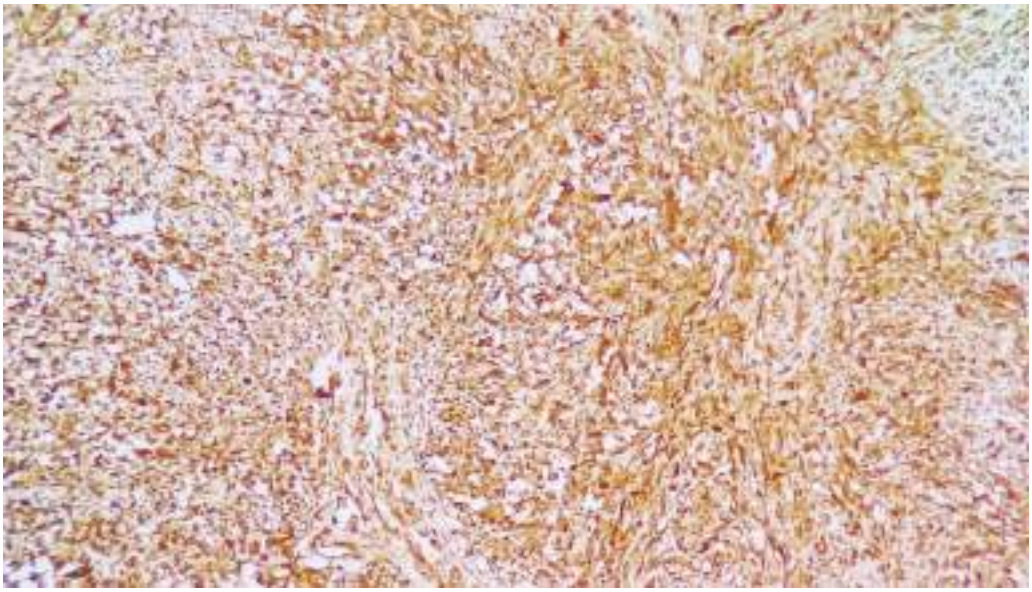


**Picture 29: Gliosarcoma – Tumor showing biphasic pattern exhibiting alternating areas of glial and mesenchymal differentiation [H&E 100X]**

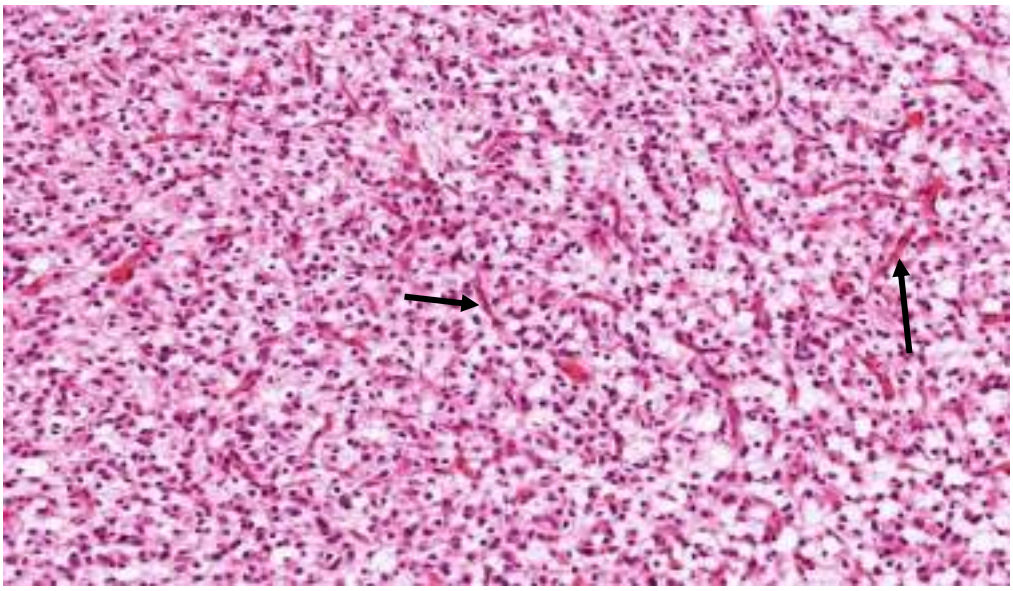
**Picture 30: Gliosarcoma – Mesenchymal spindle cell areas highlighted on reticulin stain[H&E 100X]**



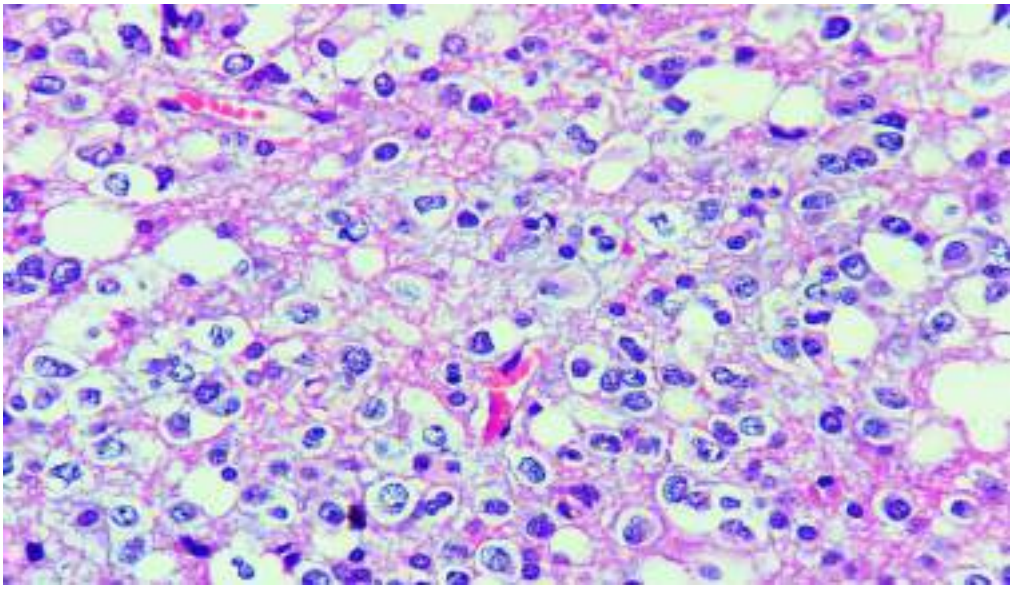
**Picture 31: Gliosarcoma – Glial comonent showing cytoplasmic GFAP immunoreactivity. [100X]**



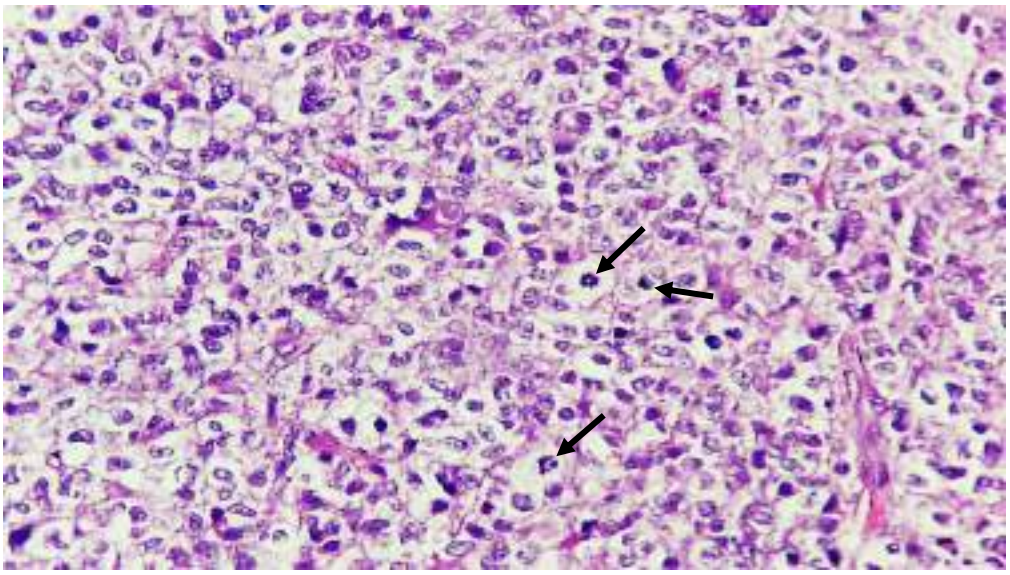
**Picture 32: Gliosarcoma - Strong cytoplasmic immunoreactivity for vimentin in mesenchymal component. [100X]**



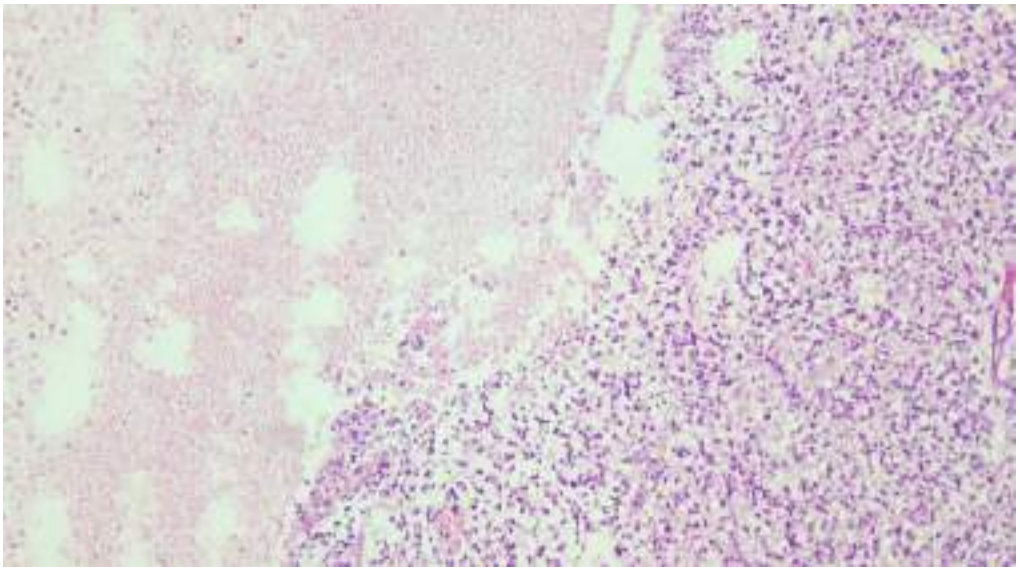
**Picture 33: Oligodendroglioma- Tumor cells arranged in sheets and have round nuclei with perinuclear halo. Chicken wire vasculature [arrow] seen. [H&E 100X]**



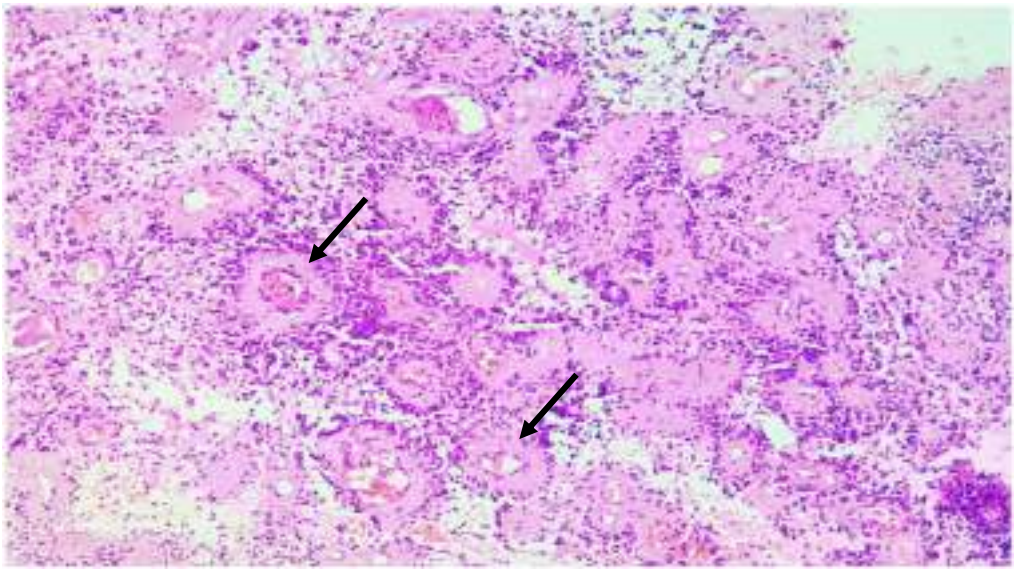
**Picture 34: Oligodendroglioma- Tumor cells are round having vesicular nuclei, perinuclear halo and moderate clear cytoplasm with well-defined cytoplasmic boundaries. [H&E 400X]**



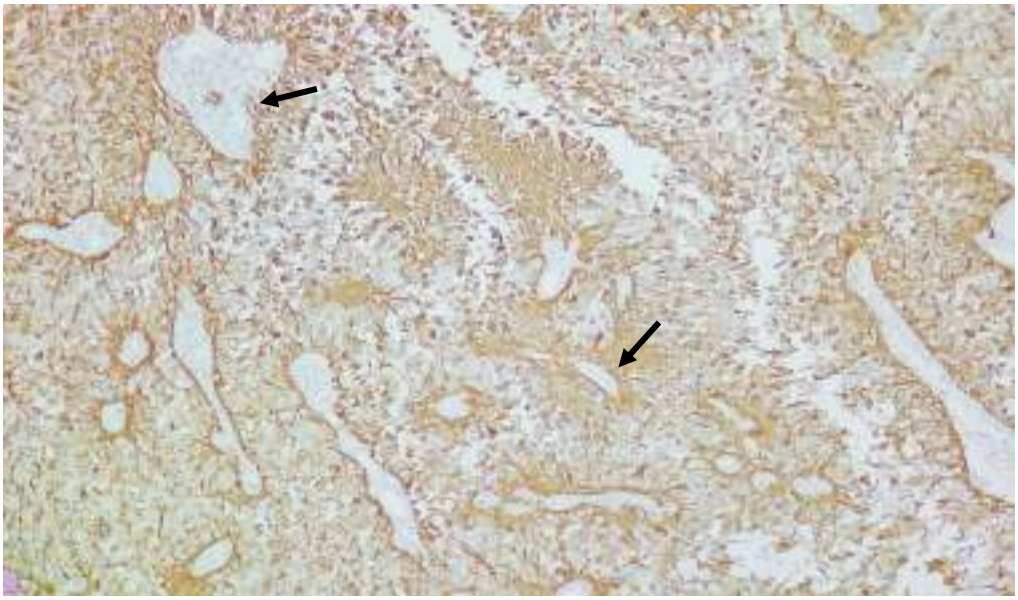
**Picture 35: Ananplastic oligodendroglioma- Tumor showing increased cellularity, nuclear hyperchromasia and increased mitotic activity [arrow]. [H&E 100X]**



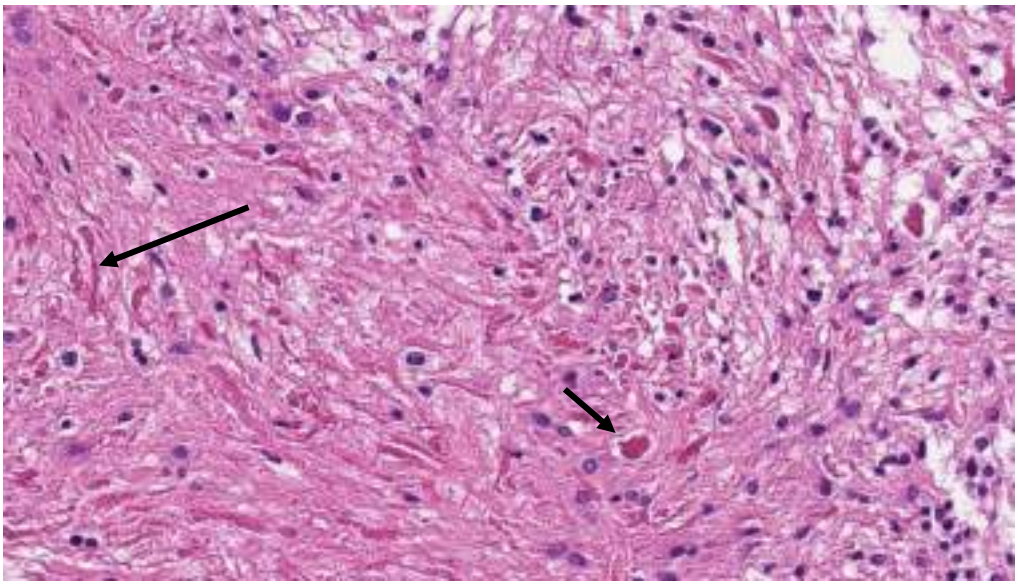
**Picture 36: Anaplastic oligodendroglioma- Tumor cells are round having vesicular nuclei, perinuclear halo and moderate amount of cytoplasm with areas of extensive necrosis. [H&E 100X]**



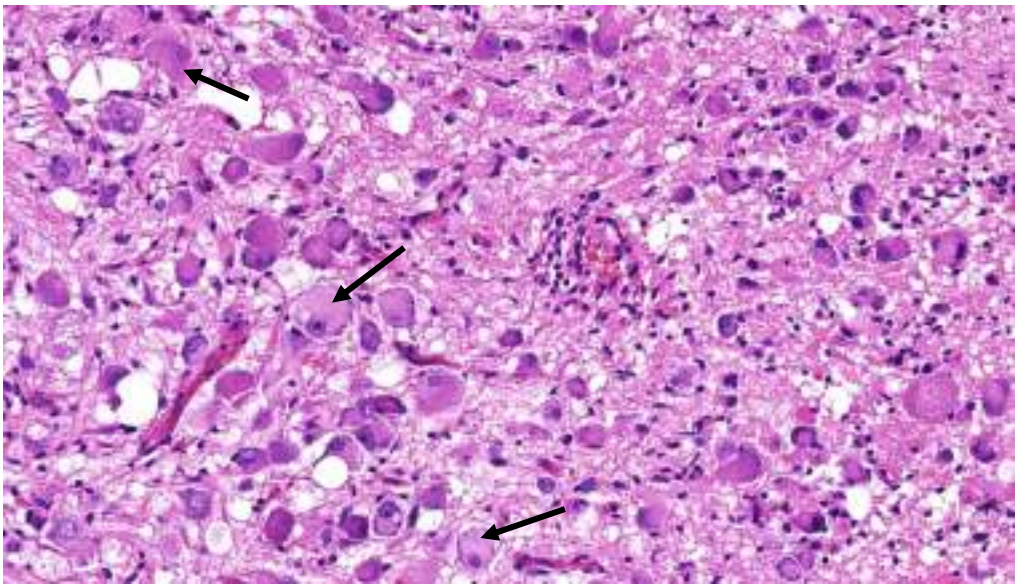
**Picture 37: Ependymoma - Perivascular Pseudo-rosettes [arrow] formed by tumor cells arranged around blood vessels forming anucleate zones. [H&E 100X]**



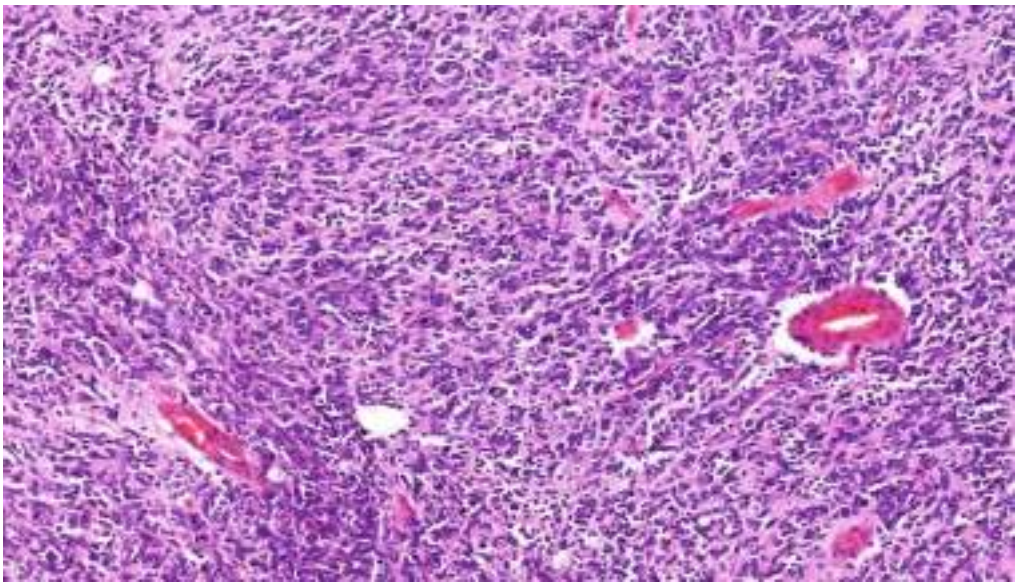
**Picture 38: Ependymoma- Tumor cells palisaded around blood vessels [arrow] show strong GFAP positivity. [cytoplasmic] [100X]**



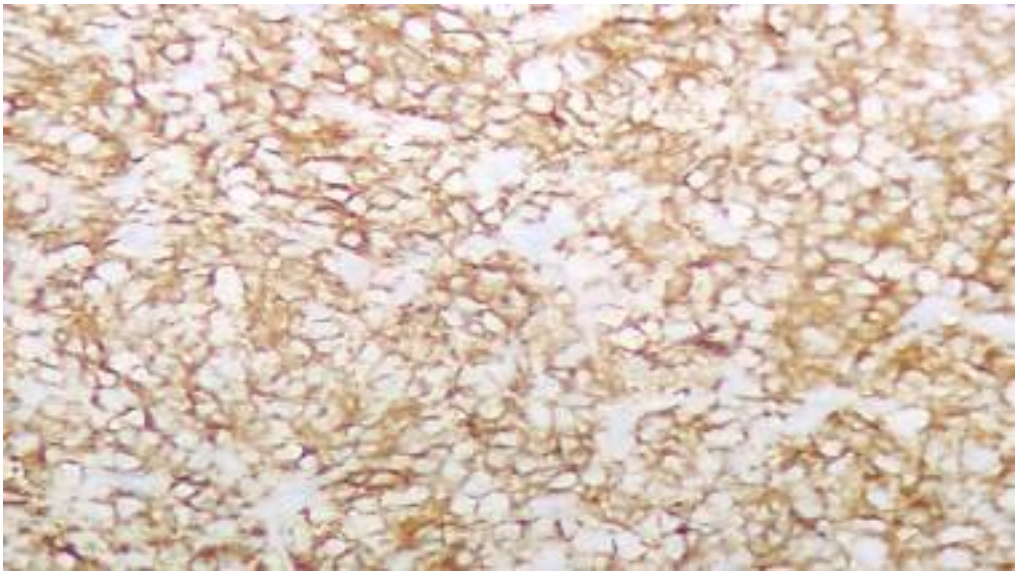
**Picture 39: Pilocytic Astrocytoma- Tumor cells in sheets with presence of numerous Rosenthal fibres [long arrow] and eosinophilic granular bodies [short arrow]. [H&E 400X]**



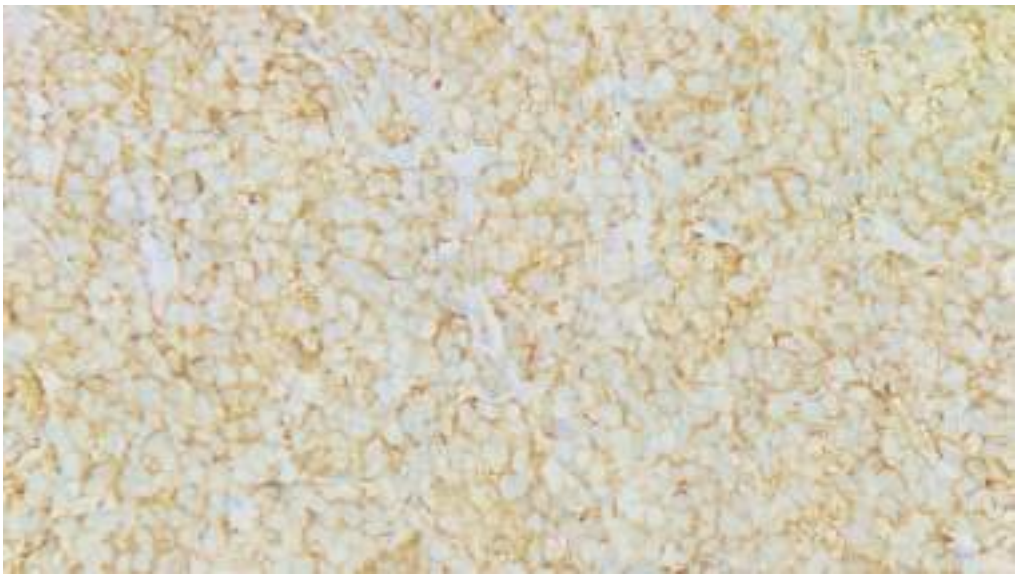
**Picture 40: Ganglioglioma- Micrograph showing biphasic pattern of irregularly oriented, dysplastic neurons [arrow] and neoplastic glial cells along with perivascular inflammatory exudates. [H&E 400X]**



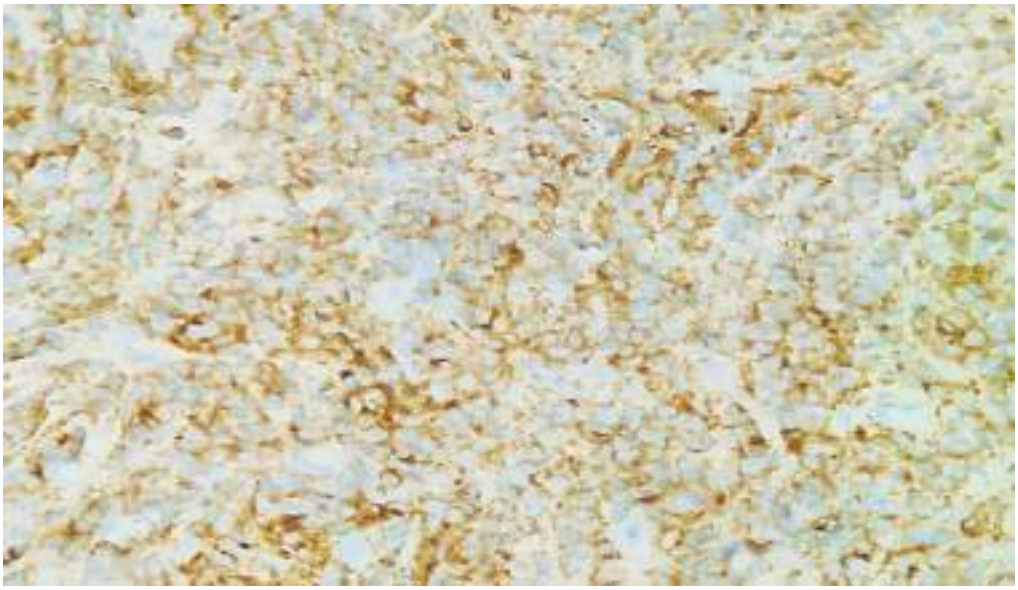
**Picture 41: Medulloblastoma- Micrograph showing round to oval hyperchromatic tumor cells arranged in diffuse sheets and cords. [H&E 100X]**



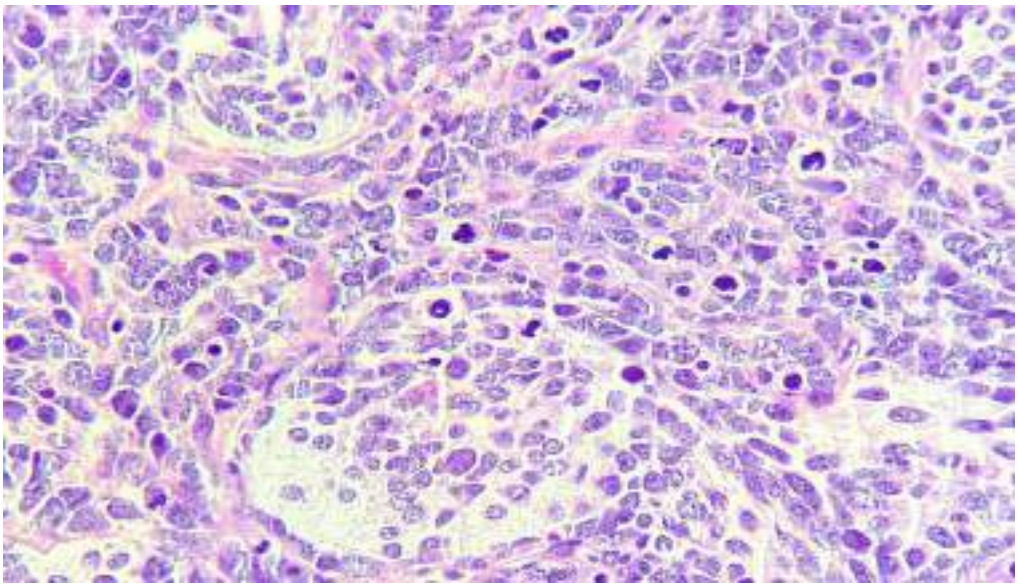
**Picture 42: Medulloblastoma- Membranous positivity of CD56. [400X]**



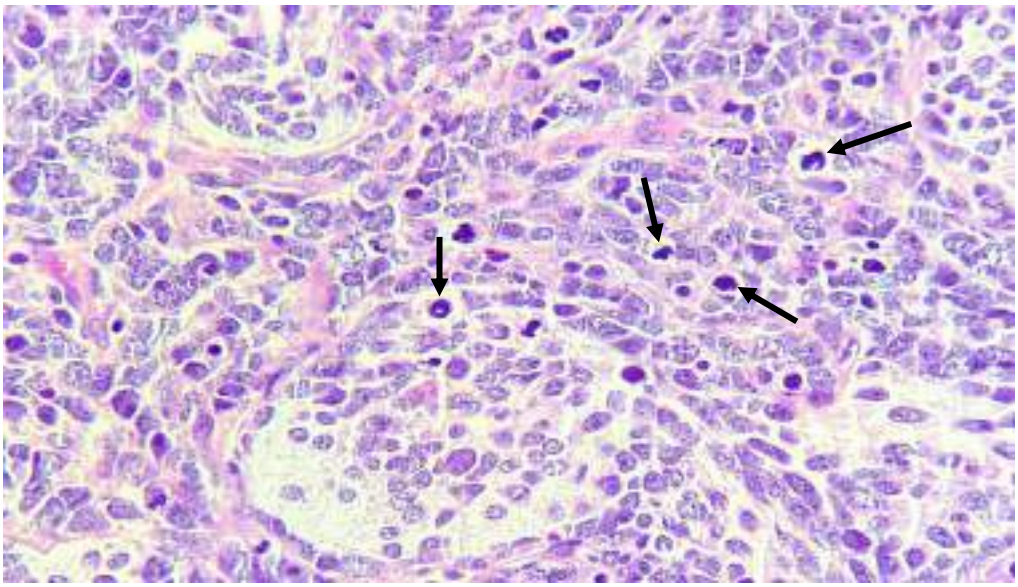
**Picture 43: Medulloblastoma- Neuron specific enolase showing membranous immunoreactivity. [100X]**



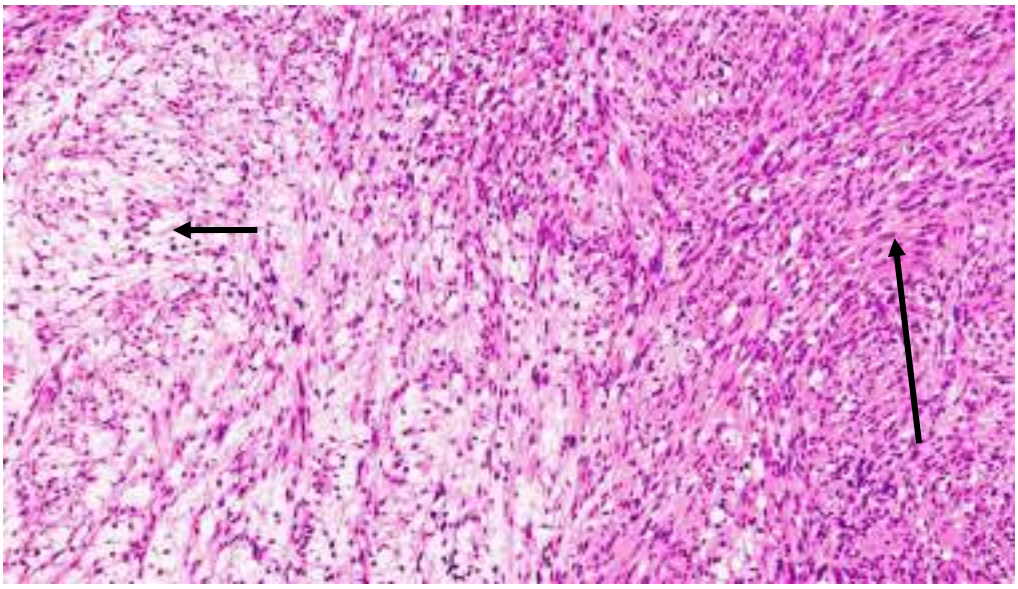
**Picture 44:Medulloblastoma- Synaptophysin showing cytoplasmic immunoreactivity. [100X]**



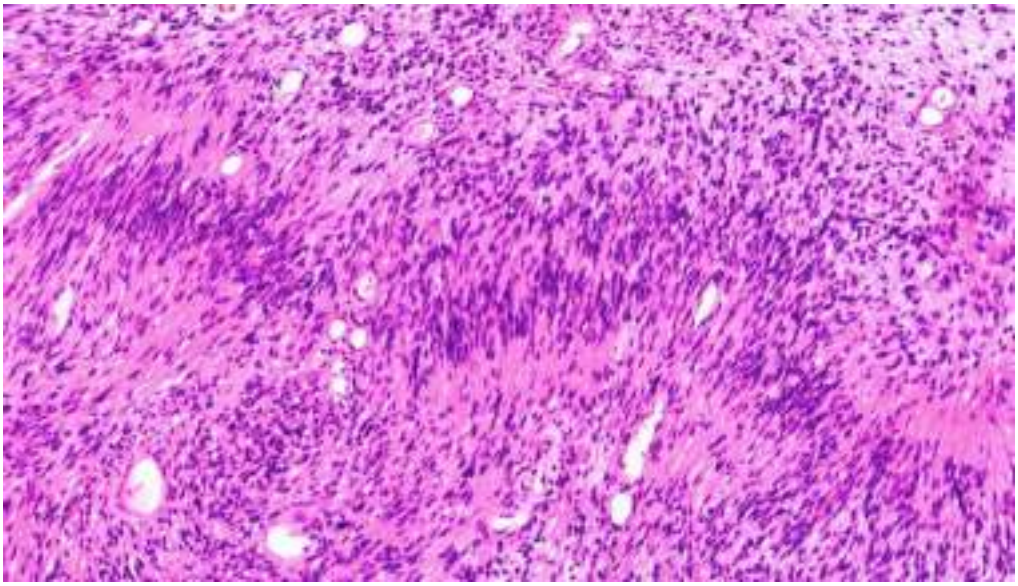
**Picture 45: Desmoplastic Medulloblastoma- Micrograph showing pale nodular areas surrounded by densely packed hyperchromatic cells. [H&E 100X]**



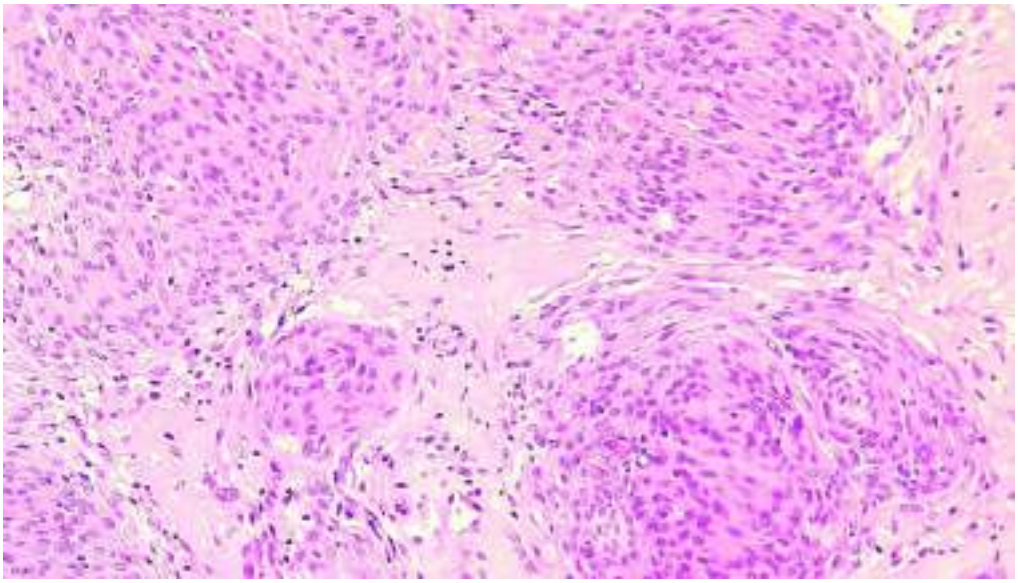
**Picture 46: Desmoplastic Medulloblastoma- Micrograph showing round to oval hyperchromatic tumor cells with brisk mitosis [arrow]. [H&E 400X]**



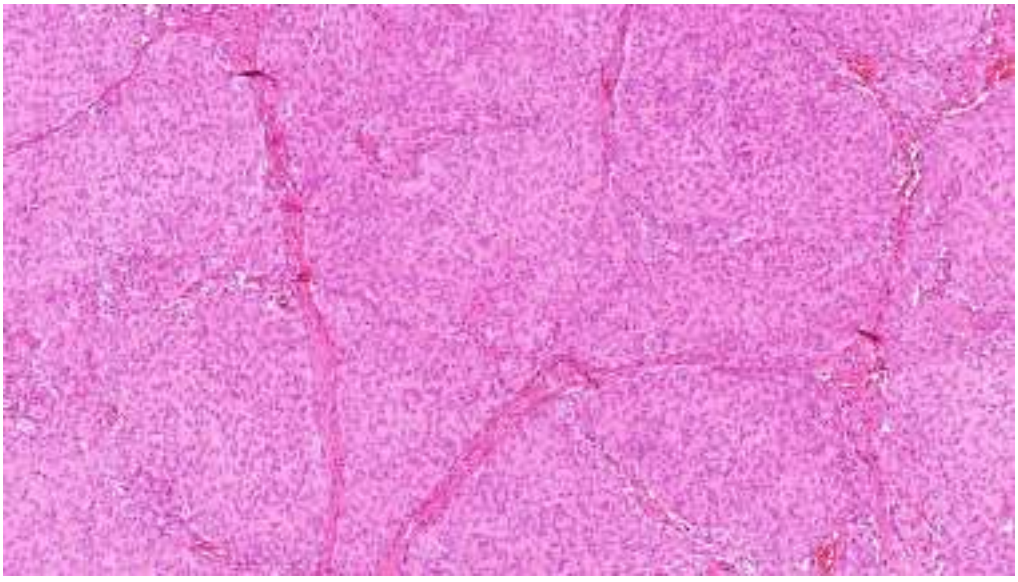
**Picture 47: Schwannoma – Cellular tumor having Antoni-A [long arrow]& Antoni-B [short arrow] areas. Cells are spindled shaped with wavy nuclei. [H&E 100X]**



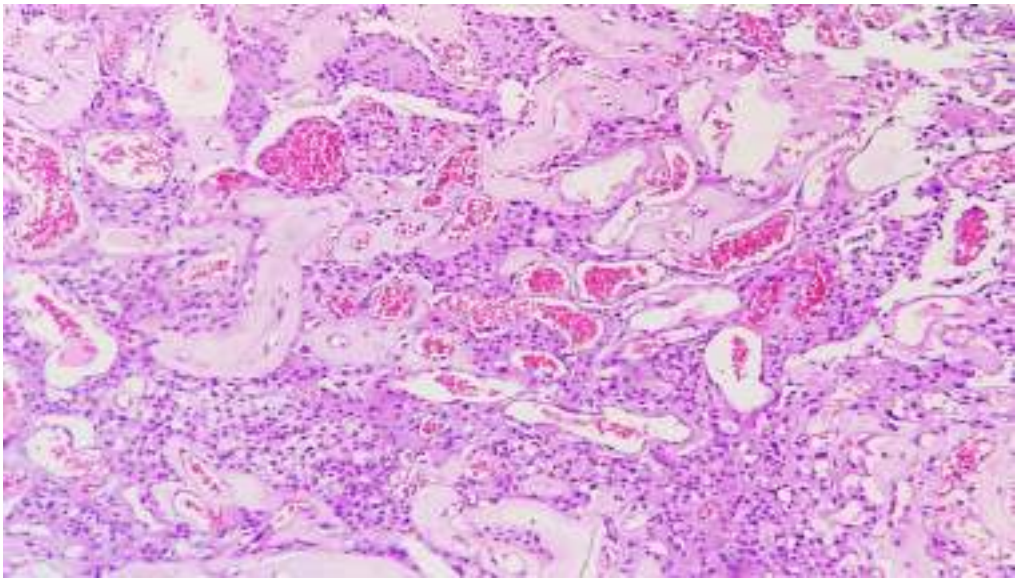
**Picture 48: Schwannoma- Verocay bodies formed by a highly ordered arrangement of Schwann cell nuclei in rows separated by fibrillary processes. [H&E 100X]**



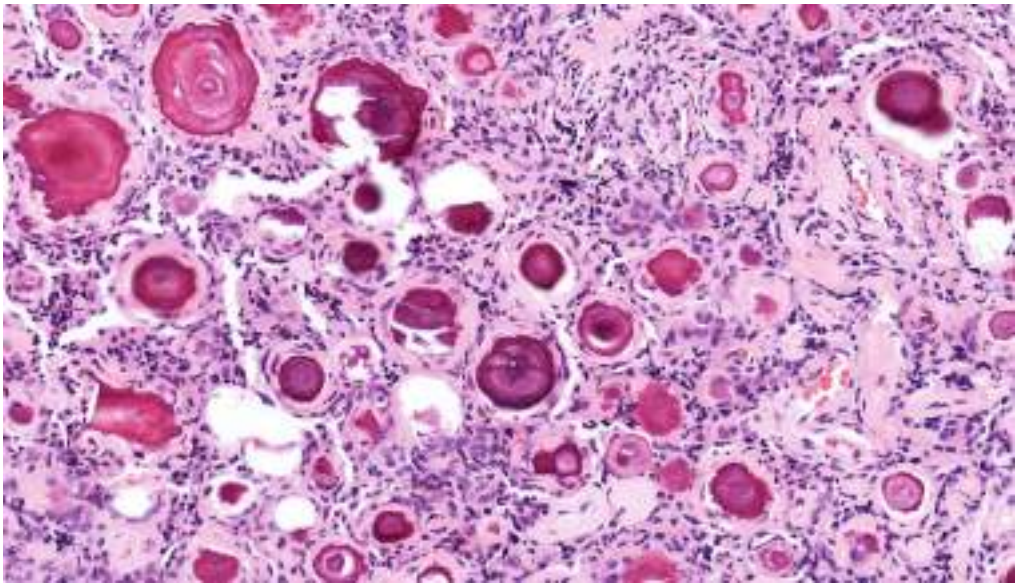
**Picture 49: Transitional Meningioma- Tumor cells arranged in whorls and fascicles. [H&E 100X]**



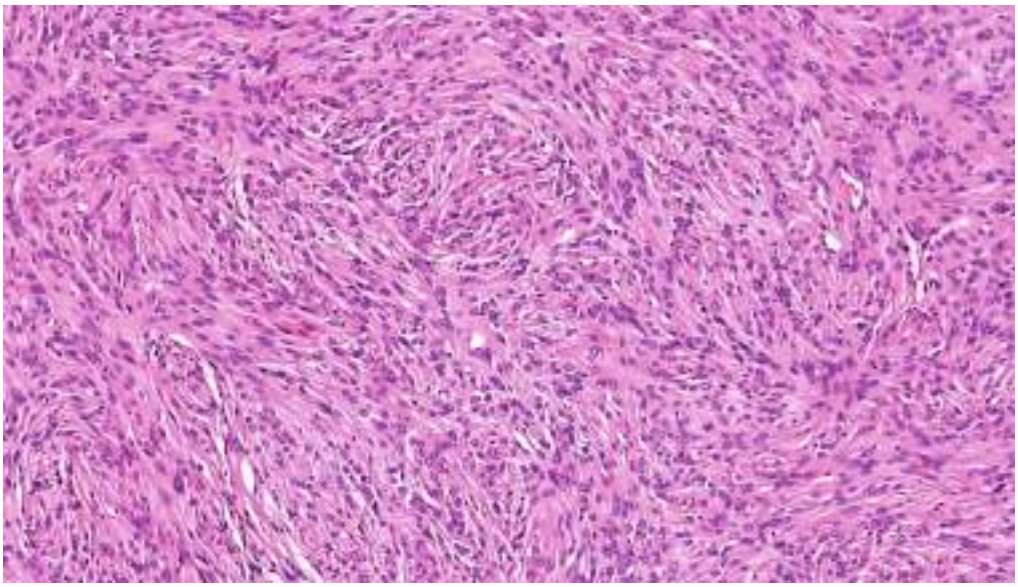
**Picture 50: Meningothelial Meningioma- Tumor cells show a lobular growth pattern, syncytium-like appearance with poorly defined cell borders. [H&E 100X]**



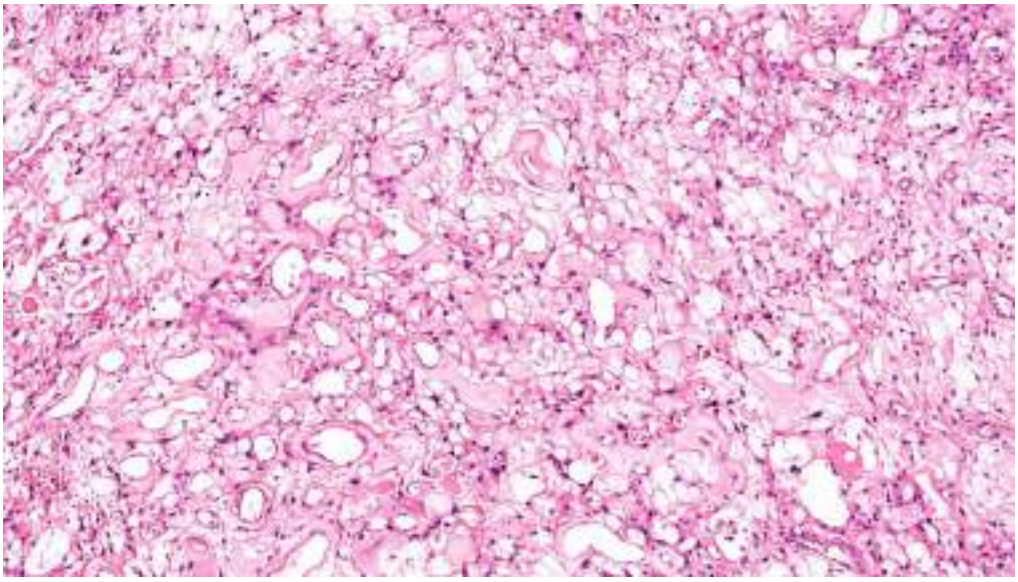
**Picture 51: Angiomatous Meningioma - Cellular tumor showing round to oval tumor cells intermingled with numerous variably sized hyalinized blood vessels. [H&E 400X]**



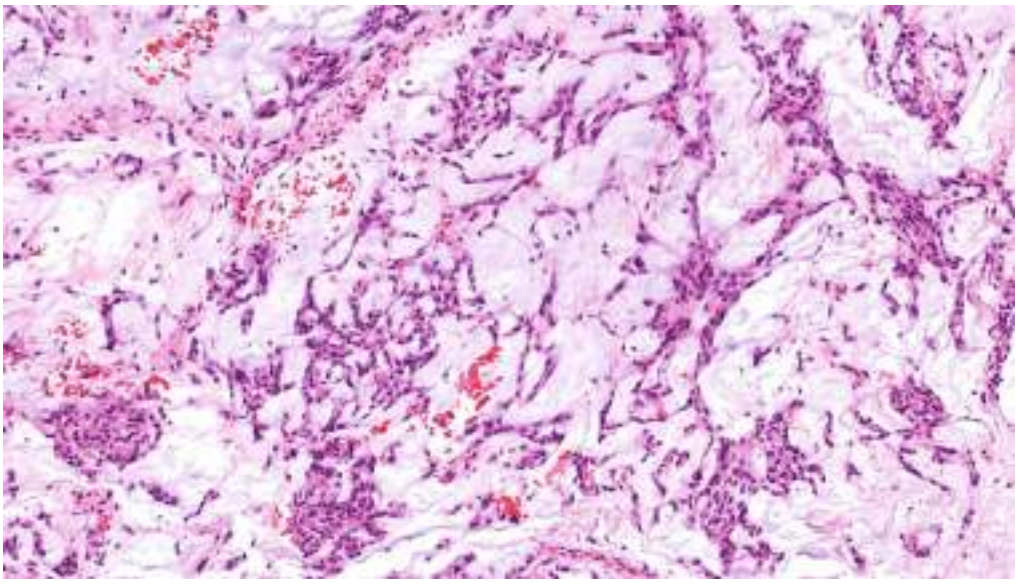
**Picture 52: Psammomatous meningioma – Prominence of psammoma bodies along with intermingled tumor cells. [H&E 400X]**



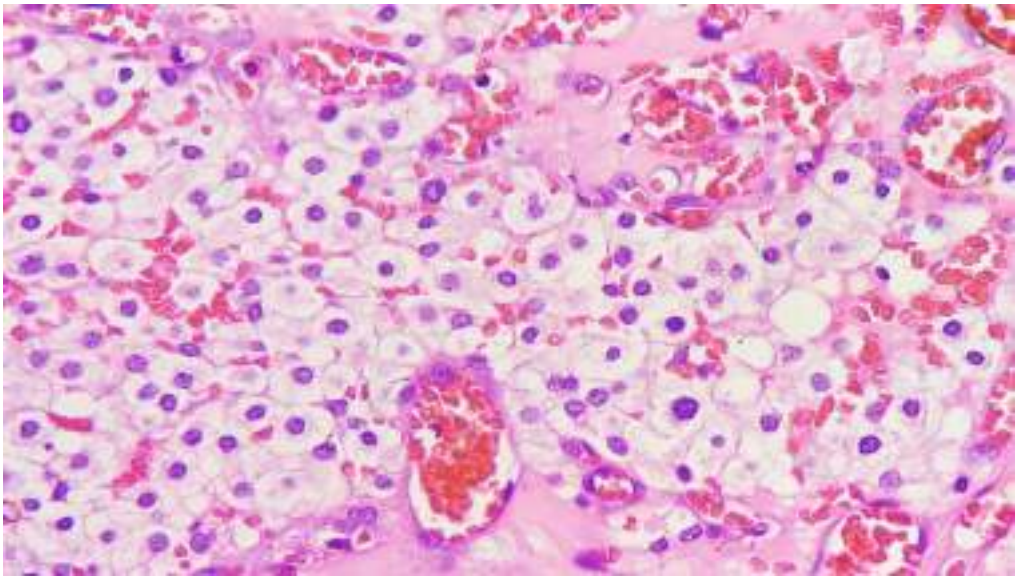
**Picture 53: Fibrous meningioma– Tumor cells form fascicles with various amounts of intercellular collagen. [H&E 100X]**



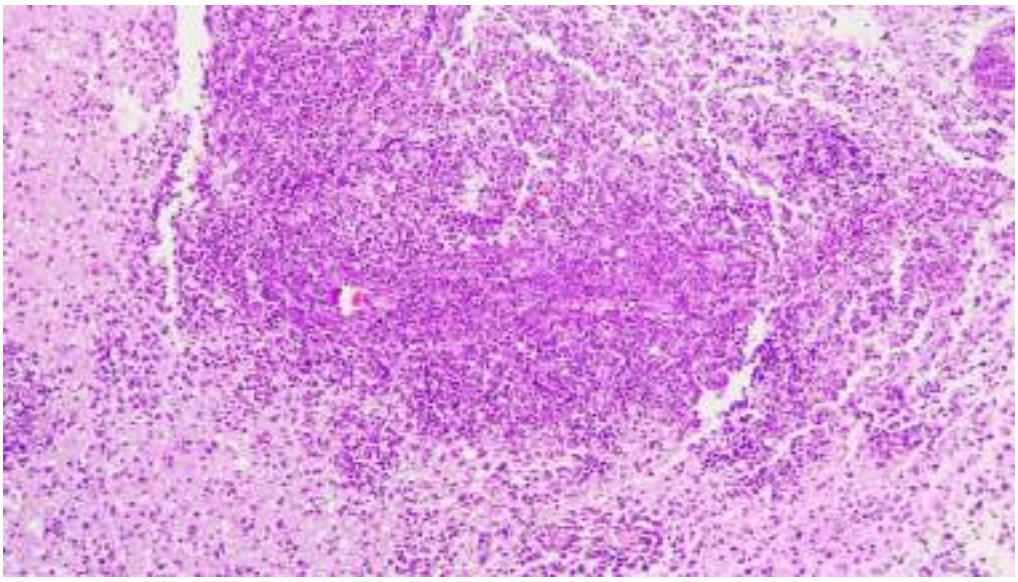
**Picture 54: Microcystic meningioma – Tumor characterized by cells with thin, elongated processes encompassing microcysts and creating a cobweb-like background. [H&E 100X]**



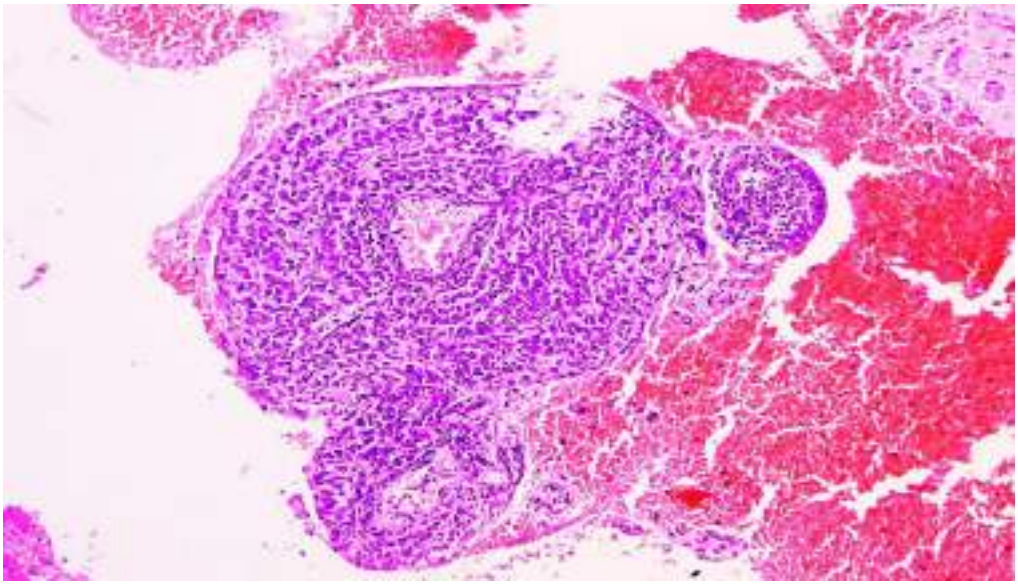
**Picture 55: Chordoid meningioma – Micrograph showing eosinophilic and vacuolated epithelioid tumor cells associated with a basophilic mucin-rich stroma. [H&E 100X]**



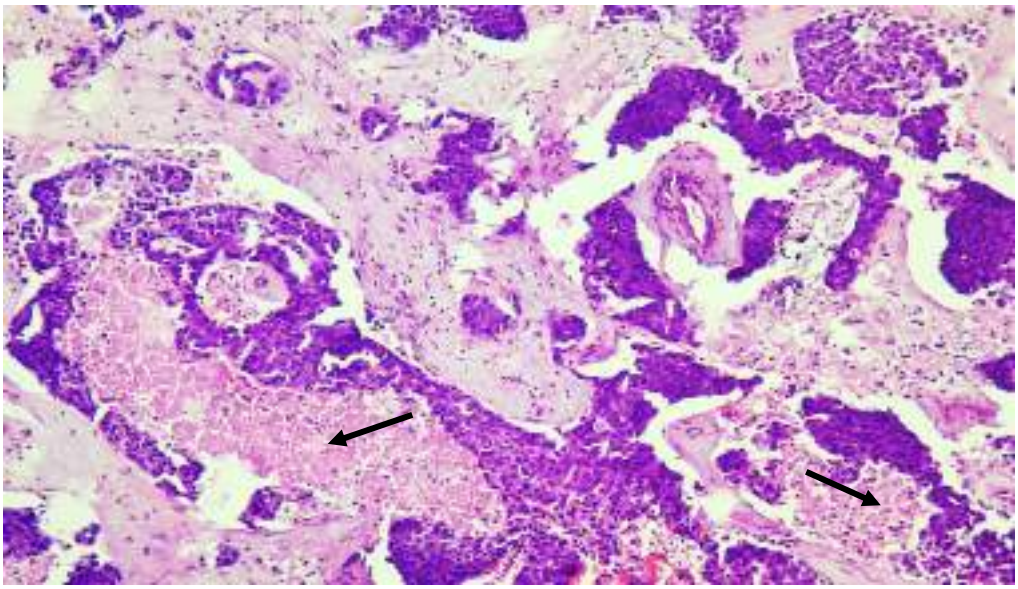
**Picture 56: Haemangioblastoma - Tumor composed of numerous thin-walled vessels surrounded by large stromal cells having vacuolated cytoplasm. [H&E 100X]**



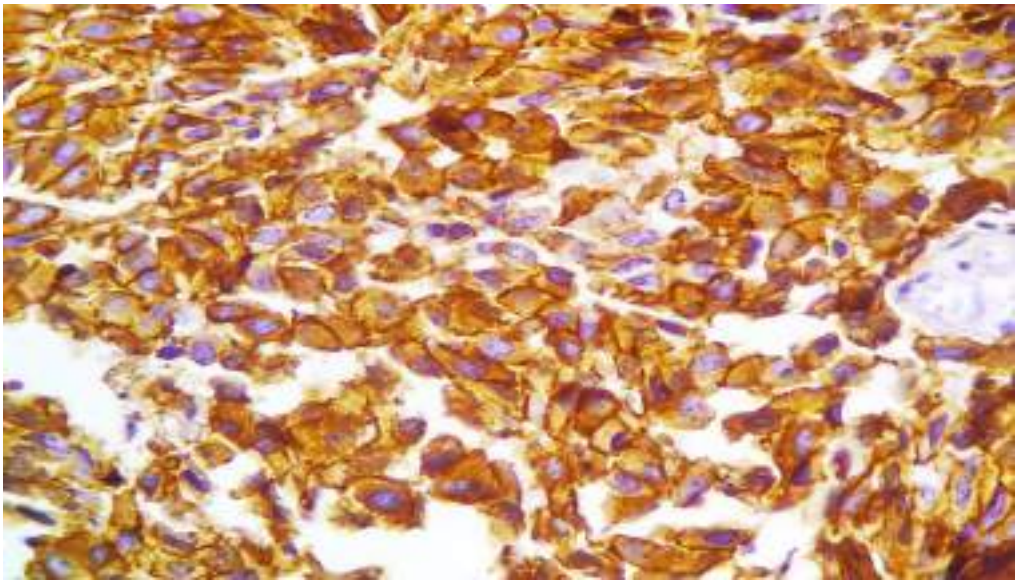
**Picture 57: Lymphoma – Brain parenchyma infiltrated by a cellular tumor with cells arranged in sheets. [H&E 100X]**



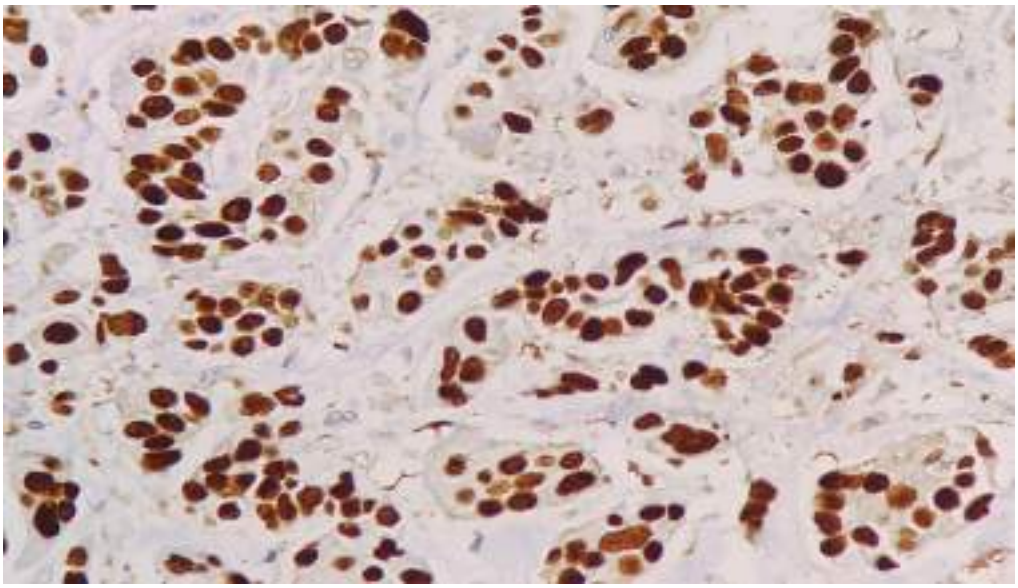
**Picture 58: Lymphoma – Expansion of blood vessel wall and dense infiltration by lymphoid cells. [H&E 400X]**



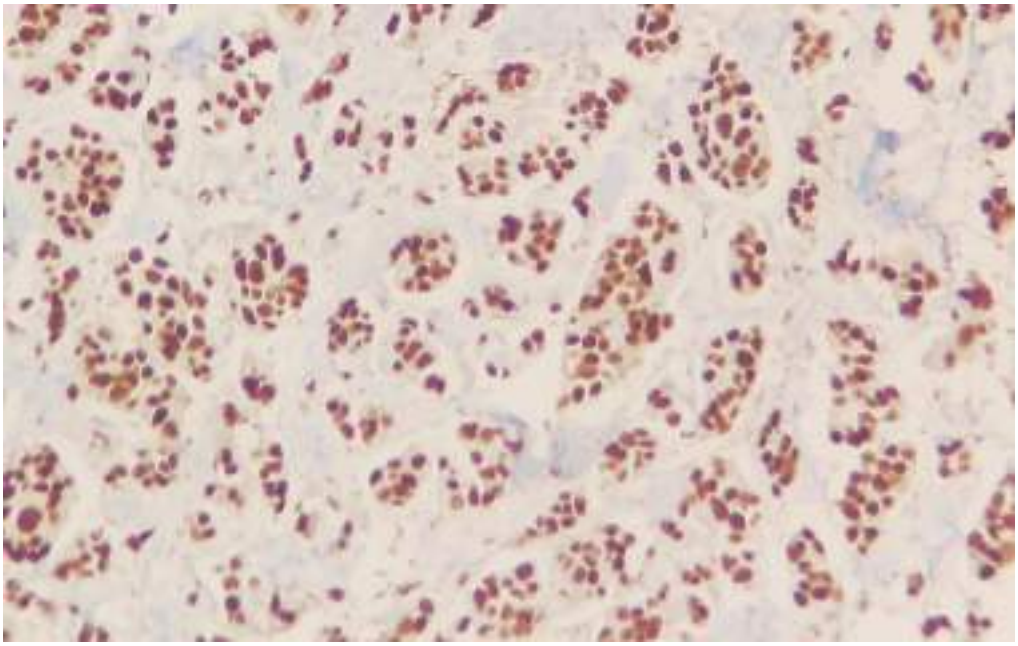
**Picture 59: Metastatic carcinomatous deposits from breast - Tumor cells arranged in nests and sheets along with focal areas of necrosis [arrow]. [H&E 100X]**



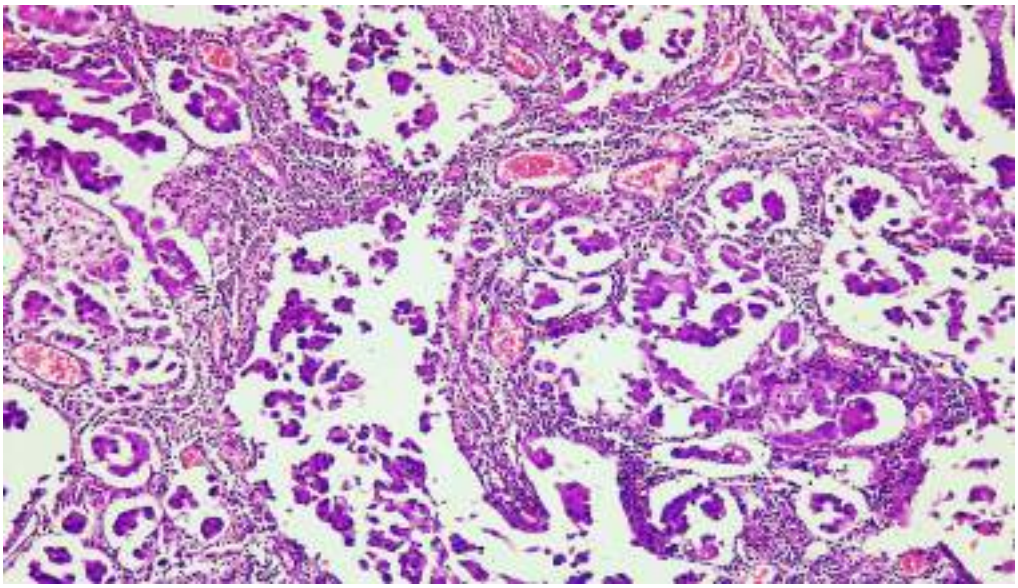
**Picture 60: Metastatic carcinomatous deposits from breast – HER2/neu positivity [membranous]. [400X]**



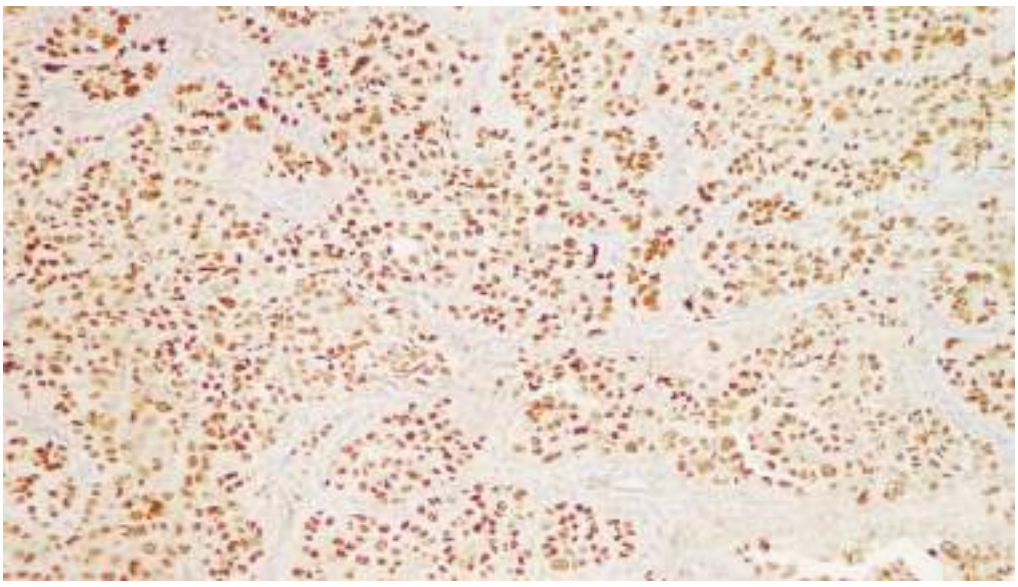
**Picture 61: Metastatic carcinomatous deposits from breast – ER positivity. [400X]**



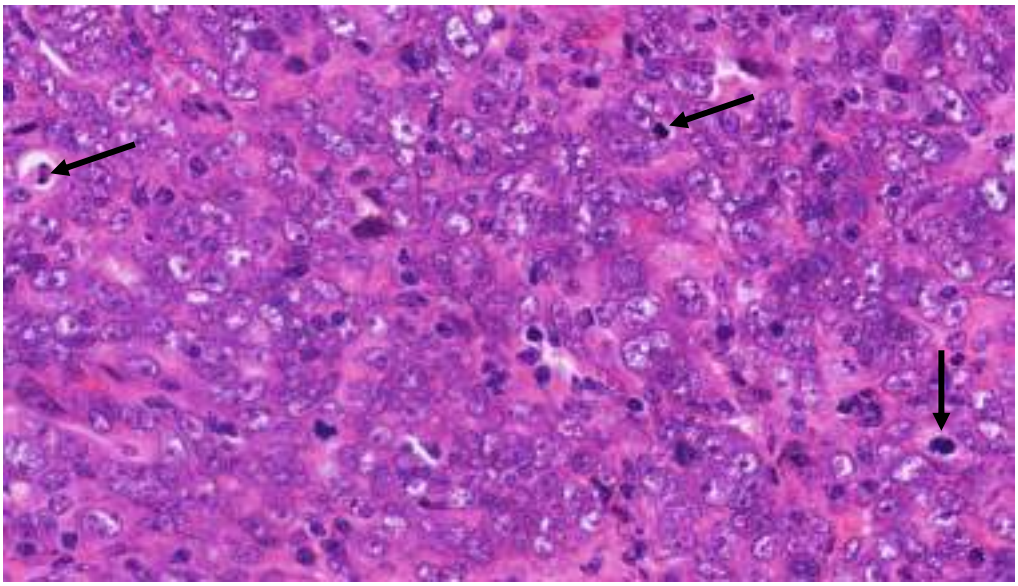
**Picture 62: Metastatic carcinomatous deposits from breast – PR positivity. [400X]**



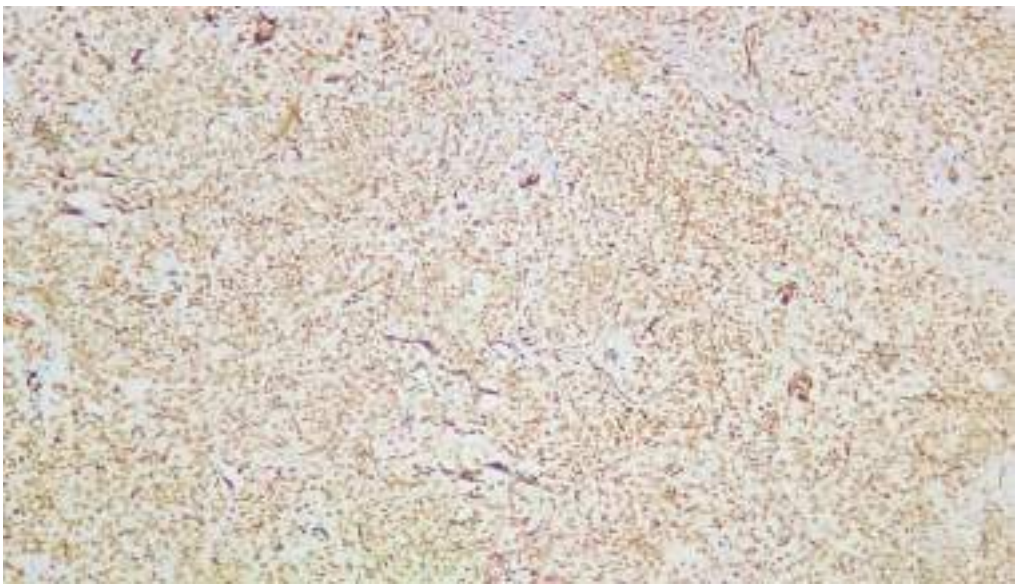
**Picture 63: Metastatic carcinomatous deposits from lung - Micrograph shows a cellular tumor cells arranged in papillae, nests and focal glands. [H&E 100X]**



**Picture 64: Metastatic carcinomatous deposits from lung – Tumor cells showing TTF1 positivity [nuclear]. [100X]**



**Picture 65: Metastatic carcinomatous deposits from ovary – Micrograph shows a cellular tumor with tumor cells arranged in nests & sheets. Nuclear atypia and mitosis noted [arrows] [H&E 400X]**



**Picture 66: Metastatic carcinomatous deposits from ovary – Tumor cells showing WT1 positivity [nuclear]. [100X]**

# KEY TO MASTERCHART

| | |
|---|---|
| M | Male |
| F | Female |
| NA | Not available |
| H/O | History of |
| K/C/O | Known case of |
| CA | Carcinoma |
| HTN | Hypertension |

| SR. NO. | MRD | HISTO NO. | NAME | AGE | SEX | CHIEF COMPLAINT | PAST HISTORY | CLINICAL DIAGNOSIS | SITE OF BIOPSY | RADIOLOGY | GROSS EXAMINATION OF SPECIMEN |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 67353 | 3176-16 | KC | 57 | M | HEADACHE & VERTIGO | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | MENINGIOMA | SINGLE 3X2X1 CM |
| 2 | 77522 | 3453-16 | RG | 50 | F | HEADACHE & SEIZURES | NA | LEFT FRONTAL GLIOMA | LEFT FRONTAL | NON ENHANCING LEISON IN LEFT FRONTAL | MULTIPLE 3.5X2.5X1 CM |
| 3 | 83867 | 3795-16 | KS | 5 | M | HEADACHE & BLURING OF VISION | NA | MEDULLOBLASTOMA | POSTERIOR FOSSA | SOLID CEREBELLAR MASS | MULTIPLE 3.5X2X1.5 CM |
| 4 | 118798 | 6641-16 | S | 28 | M | HEADACHE , MEMORY LOSS | NA | GLIOBLASTOMA | FRONTO-PARIETAL | HIGH GRADE GLIOMA | SINGLE 4.5X4.5X1 CM |
| 5 | 122689 | 7014-16 | VPS | 34 | M | HEADACHE , VERTIGO & RIGHT SIDED WEAKNESS | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 6X4X4 CM |
| 6 | 111621 | 7099-16 | MSA | 36 | M | HEADACHE , VERTIGO & RIGHT SIDED WEAKNESS | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 7.5X4X2.5 CM |
| 7 | 127683 | 7733-16 | KK | 60 | F | HEADACHE , SEIZURES & BLURING OF VISION | NA | POSTERIOR FOSSA MENINGIOMA | POSTERIOR FOSSA | MENINGIOMA | MULTIPLE 10X8X6 CM |
| 8 | 130633 | 8084-16 | SR | 57 | F | HEADACHE & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT TEMPORAL | LEFT FRONTAL HYPOINTENSE MASS | MULTIPLE 10X9X2 CM |
| 9 | 130973 | 8118-16 | DD | 58 | F | HEADACHE , LOSS OF SMELL & MEMORY LOSS | NA | OLFACTORY GROOVE MENINGIOMA | OLFACTORY GROOVE TUMOR | OLFACTORY GROOVE MENINGIOMA | MULTIPLE 6X4X2 CM |
| 10 | 133363 | 8354-16 | JS | 42 | M | HEADACHE & VERTIGO | NA | CEREBELLOPONTINE ANGLE MASS | CEREBELLOPONTINE ANGLE | BENIGN NEUROGENIC TUMOR | MULTIPLE 1X0.8X0.6 CM |
| 11 | 141564 | 9555-16 | MK | 42 | F | HEADACHE & VERTIGO | NA | RIGHT MENINGIOMA | RIGHT TEMPOTRAL | MENINGIOMA | MULTIPLE 4.5X3X2 CM |
| 12 | 138768 | 9655-16 | N | 28 | F | HEADACHE , SEIZURES & BLURING VISION | NA | LEFT MENINGIOMA | LEFT PARIETO-OCCIPITAL | MENINGIOMA | MULTIPLE 7.5X5X2 CM |
| 13 | 143469 | 9679-16 | VS | 40 | M | HEADACHE & RIGHT SIDED WEAKNESS | NA | LEFT PARIETO-OCCIPITAL SOL | LEFT PARIETO OCCIPITAL | HIGH GRADE GLIOMA | MULTIPLE 1X0.8X0.4 CM |
| 14 | 143900 | 9764/9766-16 | NK | 28 | M | HEADACHE & RIGHT SIDED WEAKNESS | NA | LEFT SUPERFICIL & DEEP TEMPORAL GLIOMA | LEFT TEMPORAL | GLIOMA | MULTIPLE 5.5X4.5X1.5 CM |
| 15 | 144018 | 9874-16 | BS | 41 | M | RIGHT SIDED WEAKNESS | NA | LEFT PARIETAL SOL | LEFT PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 1.2X1X0.3 CM |
| 16 | 149356 | 10396-16 | S | 47 | F | HEADACHE & BLURING OF VISION | NA | TUBERCULUM SELLAE SOL | TUBERCULUM SELLAE | TUBERCULUM SELLAE MENINGIOMA | MULTIPLE 1X0.8X0.6 CM |
| 17 | 16896 | 10812-16 | HS | 36 | M | HEADACHE & DIZZINESS | K/C/O RENAL CELL CARCINOMA | RIGHT FRONTAL SOL | RIGHT FRONTAL | RIGHT FRONTAL SOL | MULTIPLE 6.5X5.5X1 CM |
| 18 | 125404 | 11015-16 | S | 73 | M | HEADACHE & VERTIGO | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | MENINGIOMA | MULTIPLE 6X4X2 CM |
| 19 | 136787 | 11717-16 | SS | 16 | M | SEVERE HEADACHE & SEIZURES | NA | FOURTH VENTRICLE SOL | FOURTH VENTRICLE | FOURTH VENTRICLE SOL | MULTIPLE 4X2.5X1 CM |
| 20 | 145145 | 12193-16 | MA | 46 | M | DECREASED VISUAL ACTIVITY | NA | RIGHT SPHENOID WING MENINGIOMA | RIGHT SPHENOID WING MASS | HOMOGENEOUS ENHANCING TUMOR IN THE RIGHT SPHENOID | MULTIPLE 8X6X2 CM |
| 21 | 147199 | 12218-16 | HSG | 72 | M | HEADACHE , VERIGO & SEIZURES | NA | LEFT PARIETO-OCCIPITAL SOL | LEFT PARIETO-OCCIPITAL | MENINGIOMA | MULTIPLE 4.5X3X2.5 CM |
| 22 | 111671 | 12337-16 | HK | 55 | F | HEADACHE , SEIZURES & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | MENINGIOMA | MULTIPLE 5X5X1.5 CM |
| 23 | 152351 | 12492/12493 -16 | S | 27 | F | HEADACHE , MEMORY LOSS & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 3.5X2.5X0.8 CM |
| 24 | 154597 | 12564-16 | N | 11 | M | HEADACHE | NA | POSTERIOR FOSSA SOL | POSTERIOR FOSSA | POSTERIOR FOSSA SOL | MULTIPLE 4X3X2 CM |
| 25 | 157839 | 12693-16 | MK | 35 | F | HEADACHE & DIZZINESS | NA | LEFT PARIETAL SOL | LEFT PARIETAL | LOW GRADE GLIOMA | MULTIPLE 4.5X3.5X0.8 CM |
| 26 | 158146 | 12819-16 | RG | 43 | M | HEADACHE & SEIZURES | NA | LEFT FRONTOTEMPORAL SOL | LEFT FRONTO-TEMPORAL | HIGH GRADE GLIOMA | SINGLE 6X4X2.5 CM |
| 27 | 163971 | 13053/13054 -16 | HK | 45 | F | HEADACHE & DIZZINESS | NA | LEFT PARIETO-OCCIPITAL SOL | LEFT PARIETO-OCCIPITAL | LEFT PARIETO-OCCIPITAL SOL | MULTIPLE 4.5X4X1.5 CM |
| 28 | 173351 | 29-17 | DG | 34 | M | SEVERE HEADACHE | NA | ? MYCOTIC ANEURYSM | BRAIN VESSEL (ANEURYSM) | NA | SINGLE 1X1X0.8 CM |
| 29 | 173888 | 74-17 | MS | 68 | M | PERSONALITY CHANGES & CONFUSION | H/O CSOM | TEMPORAL ABSCESS | LEFT TEMPORAL | HYPOINTENSE RING ENHANCING LESION | MULTIPLE 3.6X2.7X1.5 CM |
| 30 | 175212 | 88-17 | AS | 62 | M | HEADACHE & WEAKNESS | H/O HEAD TRAUMA | INTRACRANIAL HEMORRHAGE | LEFT TEMPORAL | INTRACRANIL HAEMORRHAGE | MULTIPLE 2X1.5X0.8 CM |
| 31 | 174863 | 154-17 | AS | 35 | M | HEADACHE ,SEIZURES & MEMORY LOSS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 2X1.5X0.8 CM |
| 32 | 177170 | 226-17 | DK | 57 | M | HEADACHE , PERSONALITY CHANGES & WEAKNESS | NA | LEFT FRONTOTEMPORAL SOL | LEFT FRONTO-TEMPORAL | GLIOMA | MULTIPLE 3X2.5X1 CM |
| 33 | 178484 | 377-17 | SS | 19 | M | FEVER & HEADACHE | NA | ABSCESS | RIGHT TEMPORAL | NA | MULTIPLE 2.5X1.8X1.4 CM |
| 34 | 175983 | 378-17 | RK | 51 | M | HEADACHE & SEIZURES | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 2X1X0.8 CM |
| 35 | 179096 | 473-17 | VK | 54 | F | HEADACHE, SEIZURES & NEUROLOGICAL DEFICIT | K/C/O OF CA BREAST | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | METASTATIC CARCINOMA | SINGLE 2.5X2X1.5 CM |
| 36 | 183188 | 558-17 | GK | 40 | F | HEADACHE & SEIZURES | NA | LEFT TEMPOROPARIETAL SOL | LEFT TEMPORO-PARIETAL | MENINGIOMA | SINGLE 4X3X2.8 CM |
| 37 | 167340 | 573-17 | RK | 65 | F | HEADACHE & SEIZURES | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | MENINGIOMA | MULTIPLE 8.5X5X2.5 CM |
| 38 | 184118 | 678-17 | HS | 54 | M | HEADACHE,DIZZINESS & BLURING OF VISION | NA | PITUITARY MASS | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 0.5X0.3X0.2 CM |
| 39 | 184700 | 809-17 | SR | 59 | F | HEADACHE & VERTIGO | NA | SCHWANNOMA | CEREBELLOPONTINE ANGLE | BENIGN NEUROGENIC TUMOR | MULTIPLE 2.5X2X0.6 CM |
| 40 | 187900 | 827-17 | JS | 50 | M | HEADACHE,VISION DIFFICULTIES & WEAKNESS OF FACE | K/C/O HYPERTENSION | CLOTS IN BRAIN | LEFT FRONTAL | BLOOD CLOT | MULTIPLE 1.2X1X0.8 CM |
| 41 | 191139 | 1026-17 | GS | 34 | M | HEADACHE & SEIZURES | NA | BRAIN SOL | POSTERIOR FOSSA | BENIGN NEUROGENIC TUMOR | MULTIPLE 2X1.5X0.8 CM |
| 42 | 1696 | 1216-17 | S | 29 | F | HEADACHE & SEIZURES | NA | LEFT FRONTAL MENINGIOMA | LEFT FRONTAL | MENINGIOMA | MULTIPLE 6X5X4 CM |
| 43 | 193975 | 1246-17 | DKJ | 58 | M | HEADACHE & SEIZURES | NA | RIGHT OCCIPITAL SOL | RIGHT OCCIPITAL | LOW GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 44 | 198641 | 1485-17 | VS | 65 | M | HEADACHE & DIZZINESS | K/C/O RENAL CELL CARCINOMA | CEREBELLAR SOL | CEREBELLUM | HYPOINTENSE MASS | MULTIPLE 3X2.5X2 CM |
| 45 | 197185 | 1503-17 | HK | 25 | M | HEADACHE | NA | LEFT FRONTO-TEMPORAL MASS | LEFT FRONTO-TEMPORAL | GLIOMA | SINGLE 3X2X1 CM |
| 46 | 187539 | 1521-17 | GS | 56 | M | HEADACHE | NA | LEFT PARIETAL SOL | LEFT PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 5X3.5X2 CM |
| 47 | 202394 | 1693-17 | MC | 55 | M | HEADACHE & BLURING OF VISION | NA | RIGHT TEMPORAL SOL | RIGHT TEMPOTAL | MASS RIGHT TEMPORAL | MULTIPLE 5.5X4X3 CM |
| 48 | 202931 | 1841-17 | PK | 55 | F | HEADACHE | NA | MENINGIOMA | LEFT TEMPORO-PARIETAL | MENINGIOMA | MULTIPLE 1.7X1X0.8 CM |
| 49 | 207921 | 1899-17 | RS | 28 | M | BACK PAIN & LOCALIZED PAIN | NA | INTRADURAL EXTRAMEDULLARY SOL | RIGHT OCCIPITAL | BENIGN NEUROGENIC TUMOR | MULTIPLE 2X1X1 CM |
| 50 | 206237 | 1900-17 | AS | 27 | M | HEADACHE & BLURING OF VISION | NA | RIGHT PARIETAL SOL | RIGHT PARIETAL | ASTROCYTOMA | MULTIPLE 1.5X1.5X1 CM |
| 51 | 208396 | 1901-17 | AS | 52 | M | HEADACHE | NA | PARIETO -OCCIPITAL SOL | LEFT PARIETO-OCCIPITAL | GLIOMA | MULTIPLE 1X0.5X0.5 CM |
| 52 | 61337 | 1995-17 | KG | 50 | F | HEADACHE,SEIZURS & BLURING OF VISION | NA | CEREBRAL SOL | RIGHT FRONTAL | MENINGIOMA | MULTIPLE 6X4X2 CM |
| 53 | 86271 | 2032-17 | SS | 24 | M | HEADACHE,SEIZURES & MEMORY LOSS | NA | TEMPORAL SOL | RIGHT TEMPORAL | SUPRATENTORIAL LESION | MULTIPLE 5X3X1 CM |
| 54 | 197175 | 2061-17 | RS | 18 | M | HEADACHE,ABSENSE OF SMELL & WEAKNESS OF WHOLE BODY | NA | FRONTAL ABSCESS | LEFT FRONTAL | BRAIN ABSCESS | MULTIPLE 1.5X1X0.8 CM |
| 55 | 34673 | 2062-17 | JS | 32 | M | HEADACHE WITH WEAKNESS | K/C/O GERM CELL TUMOR | FRONTAL SOL | RIGHT FRONTAL | METASTATIC CARCINOMA | MULTIPLE 6X5X4 CM |
| 56 | 181419 | 2119-17 | HS | 49 | M | HEADACHE & DIZZINESS | K/C/O RENAL CELL CARCINOMA | METASTATIC MALIGNANCY | LEFT FRONTAL | METASTATIC CARCINOMA | MULTIPLE 5.5X4X2.8 CM |
| 57 | 214430 | 2164-17 | PB | 39 | M | HEADACHE & VERTIGO | NA | CEREBELLOPONTINE ANGLE SOL | CEREBELLOPONTINE ANGLE | BENIGN NEUROGENIC TUMOR | MULTIPLE 3.5X3X0.8 CM |
| 58 | 210811 | 2319-17 | MK | 29 | F | HEADACHE & BLURING OF VISION | NA | BRAIN SOL | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 1.5X1X0.5 CM |
| 59 | 142918 | 2420-17 | SD | 66 | F | HEADACHE,VERTIGO & BLURING OF VISION | NA | BRAIN SOL | LEFT TEMPORAL | MENINGIOMA | MULTIPLE 6X5X1.5 CM |
| 60 | 219156 | 2463-17 | HS | 46 | M | HEADACHE,LOSS OF SMELL & PERSONALITY CHANGES | NA | FRONTAL SOL | RIGHT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 3.5X3X1.5 CM |
| 61 | 219841 | 2465-17 | BS | 60 | M | HEADACHE,DIZZINESS & WEAKNESS OF FACE | NA | BLOOD CLOT IN BRAIN | LEFT TEMPORAL | INTRACRANIL HAEMORRHAGE | MULTIPLE 5X4X2.5 CM |
| 62 | 221657 | 2616-17 | JK | 42 | F | HEADACHE,MEMORY LOSS & BLURING OF EYES | NA | BRAIN SOL | RIGHT OCCIPITAL | MENINGIOMA | MULTIPLE 3.5X3X1.2 CM |
| 63 | 203073 | 2671-17 | KK | 59 | M | HEADACHE & BLURING OF VISION | NA | RIGHT PARIETO-OCCIPITAL SOL | RIGHT PARIETO OCCIPITAL | GLIOMA | MULTIPLE 1.2X0.7X0.4 CM |
| 64 | 221526 | 2732-17 | KR | 50 | M | HEADACHE | NA | FRONTAL SOL | LEFT FRONTAL | MENINGIOMA | MULTIPLE 9X7X3 CM |
| 65 | 221014 | 2841-17 | JS | 28 | M | HEADACHE & VISION LOSS | NA | FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 66 | 128564 | 2985-17 | M | 44 | F | HEADACH & BLURING OF VISION | NA | LEFT PARIETAL SOL | LEFT PARIETAL | MENINGIOMA | MULTIPLE 2X1.5X0.8 CM |
| 67 | 231442 | 3139-17 | MK | 43 | F | HEADACHE,VISION PROBLEM & MOOD CHANGES | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 5X2X1.5 CM |
| 68 | 203389 | 3193-17 | GK | 59 | F | HEADACHE,VISION CHANGES & MOOD CHANGES | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 2X1X0.8 CM |
| 69 | 23086 | 3612-17 | VS | 40 | M | HEADACHE,VISION LOSS & MEMORY LOSS | NA | LEFT FRONTO-TEMPORAL SOL | LEFT FRONTO-TEMPORAL MASS | GLIOMA | MULTIPLE 2X1.8X0.4 CM |
| 70 | 240675 | 3725-17 | PS | 42 | M | HEADACHE | NA | RIGHT PARASAGGITAL SOL | RIGHT PARASAGGITAL | MENINGIOMA | MULTIPLE 3.5X2X1.5 CM |
| 71 | 240753 | 3760-17 | MS | 21 | M | HEADACHE & HEARING LOSS | NA | INTRADURAL EXTRAMEDULLARY LESION | LEFT TEMPORO-OCCIPITAL | BENIGN NEUROGENIC TUMOR | SINGLE 2X1.5X1.5 CM |
| 72 | 242845 | 3995-17 | DR | 47 | M | HEADACHE & PROBLEMS IN MEMORY | NA | BRAIN SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 73 | 247484 | 4101-17 | MK | 75 | F | HEADACHE & WEAKNESS | NA | INTRACRANIAL HEMORRHAGE | RIGHT TEMPORO-PARIETAL | INTRACRANIAL HAEMORRHAGE | MULTIPLE 0.8X0.5X0.2 CM |
| 74 | 260907 | 5030-17 | SS | 47 | M | MEMORY LOSS,LOSS OF SMELL & SEIZURES | NA | RIGHT TEMPORAL LESION | RIGHT TEMPORAL | GLIOMA | MULTIPLE 7X5X3 CM |
| 75 | 272283 | 6024-17 | RK | 45 | M | FEVER & HEADACHE | NA | RIGHT FRONTAL ABSCESS | RIGHT FRONTAL | ABSCESS | MULTIPLE 8X6X3 CM |
| 76 | 278682 | 6669-17 | HK | 30 | F | HEADACHE | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 2X1X0.5 CM |
| 77 | 289978 | 7270-17 | MI | 48 | M | SEIZURES & SHORT TERM MEMORY LOSS | NA | RIGHT TEMPORAL TUMOR | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 3X2X1 CM |
| 78 | 293837 | 7735-17 | GK | 37 | F | HEADACHE,VOMITING & DIFFICULTY IN WALKING | NA | CEREBELLAR TUMOR | RIGHT CEREBELLUM | MENINGIOMA | MULTIPLE 5X4X2.5 CM |
| 79 | 320492 | 9058-17 | PW | 43 | M | HEADACHE & IMBALANCE WHILE WALKING | NA | TUMOR OF POST FOSSA | POSTERIOR FOSSA | POSTERIOR FOSSA SOL ?MENINGIOMA | MULTIPLE 15X11X6 CM |
| 80 | 339045 | 10263-17 | SK | 49 | F | HEADACHE & NEUROLOGICAL DEFICIT | NA | LEFT FRONTAL SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 8X6X2 CM |
| 81 | 341494 | 10466-17 | HS | 21 | M | HEADACHE & SEIZURES | NA | ARTERIO-VENOUS MALFORMATION | LEFT FRONTAL | NA | SINGLE 1X0.5X0.5 CM |
| 82 | 347512 | 10790-17 | SK | 6 | F | HEADACHE & VOMITING | NA | CEREBELLAR TUMOR | LEFT CEREBELLUM | LOW GRADE GLIOMA | MULTIPLE 5X3X1.8 CM |
| 83 | 357669 | 11552-17 | NK | 26 | F | HEADACHE & UNCOORDINATED MUSCLE MOVEMENT ATAXIA | NA | RIGHT CEREBELLAR SOL | RIGHT CEREBELLUM | HYPOINTENSE LESION | MULTIPLE 4X3X2 CM |
| 84 | 360383 | 11845-17 | SK | 48 | F | HEADACHE & BLURING OF VISION | K/C/O CA OVARY | BRAIN SOL | RIGHT PARIETAL | METASTATIC CARCINOMA | MULTIPLE 2X2X1 CM |
| 85 | 373685 | 12434-17 | KK | 58 | F | HEADACHE & VOMITING | NA | RIGHT CEREBELLAR SOL | RIGHT CEREBELLAR | MENINGIOMA | MULTIPLE 9X5X1.5 CM |
| 86 | 374039 | 12437-17 | KR | 64 | M | HEADACHE & VOMITING | NA | RIGHT PARIETAL SOL | RIGHT PARIETAL | NA | MULTIPLE 9X6X4.5 CM |
| 87 | 378279 | 12720-17 | SD | 65 | F | HEADACHE & RIGHT SIDED WEAKNESS | NA | BRAIN LEFT FLAX SOL | LEFT PARIETAL | MENINGIOMA | SINGLE 3X2.5X1 CM |
| 88 | 388390 | 13366-17 | P | 40 | M | HEADACHE,DIZZINESS & BLURING OF VISION | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 4X3.5X2 CM |
| 89 | 391144 | 13414-17 | AS | 2 | M | HEADACHE & VOMITING | NA | EXTRADURAL TISSUE | EXTRADURAL TISSUE | DERMOID CYST | MULTIPLE 2X1.5X0.1 CM |
| 90 | 173351 | 13530-16 | DG | 34 | M | HYPERTENSION & MUCAL RIGIDITY | H/O HEAD TRAUMA | INTRACRANIAL HEMATOMA | RIGHT TEMPORAL | THROMBOSIS | MULTIPLE 1X0.8X0.5 CM |
| 91 | 396053 | 13794-17 | LS | 59 | M | HEADACHE & WEAKNESS | NA | LEFT PARIETAL SOL | LEFT PARIETAL | LEFT PARIETAL MASS | MULTIPLE 3X1.5X1.2 CM |
| 92 | 398025 | 14084-17 | SK | 43 | F | HEADACHE,DIZZINESS & BLURING OF VISION | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 2X1.5X0.6 CM |
| 93 | 400904 | 14086-17 | BS | 55 | M | HEADACHE,DIZZINESS & WEAKNESS | NA | INTRACRANIAL HEMORRHAGE | LEFT FRONTAL | THROMBOSIS | MULTIPLE 6X4X2 CM |

| SR. NO. | MRD | MICROSCOPIC EXAMINATION | SPECIAL STAINS | | | | |
|---|---|---|---|---|---|---|---|
| | | | AFB | PAS | GMS | RETICULIN | PERL'S |
| 1 | 67353 | SHOW A CELLULAR TUMOR WITH VARIABLY SIZED HYALINISED BLOOD VESSELS. TUMOR CELLS ARE ROUND TO OVAL WITH INDISTINCT CELL BOUNDARIES, FINE NUCLEAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. OCCASIONAL LARGE CELLS WITH HIGH N:C RATIO SEEN. NO ITOSIS/NECROSIS/SHEETING PATTERN SEEN. NO NORMAL BRAIN PARENCHYMA | NA | NA | NA | NA | NA |
| 2 | 77522 | NDIVIDUAL TUMOR HAVE ROUND VESICULAR NUCLEI, ABUNDANT AMOUNT OF CLEAR CYTOPLASM- OLIGODENDROGIAL COMPONENT PRESENT AGAINST CHICKEN WIRE VASCULATURE . ALSO SEEN ARE PLEOMORPHIC HYPERHCROMATIC NUCLEI AGAINST A FIBRILLARY BACKGROUND ASTROCYTIC COMPONENT. ATYPICAL MITOSIS SEEN. LARGE AREA OF NECROSIS PA | NA | NA | NA | NA | NA |
| 3 | 83867 | SECTIONS SHOW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN CORDS AND SHEETS HAVING HYPERCHROMATIC NUCLEI, INCONSPICUOUS NUCLEOLI AND SCANT AMOUNT OF CYTOPLASM. THESE CELLULAR AREAS ARE SURROUNDING THE NODULAR PALE ISLANDS WHICH HAVE REDUCED CELLULARITY AND A FIBRILLARY MATRIX. | NA | NA | NA | NA | NA |
| 4 | 118798 | DIFFUSE SHEETS. THE INDIVIDUAL TUMOR CELLS ARE HIGHLY PLEOMORPHIC HAVE HYPERCHROMATIC NUCLEI AGAINST A FIBRILLARY BACKGROUND. NUMEROUS MULTINUCLEATED GIANT CELLS IDENTIFIED. AREAS OF NECROSIS AND ENDOTHELIAL CELL PROLIFERATION ALONG WITH PRESENCE OF AN OCCASIONAL GLOMERULOID STRUCTURE IDENTIFIED. MITOTIC | NA | NA | NA | NA | NA |
| 5 | 122689 | USE SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI INCONSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. GEMISTOCYTES, MICROCYSTIC CHANGE, COLLECTIONS OF FOAMY MACROPHAGES AND FOCAL MYXOID CHANGE.NOTED.NECROSIS AND ENDOTHELIAL CELL PROLIFERATION SEEN. NO | NA | NA | NA | NA | NA |
| 6 | 111621 | TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS AGAINST FIBRILLARY BACKGROUND. THE TUMOR CELLS ARE PLEOMORPHIC AND HAVE HYPERCHROMATIC NUCLEI. ATYPICAL MITOTIC FIGURES, ENDOTHELIAL PROLIFERATION AND AREA OF NECROSIS SEEN. FEW GEMISTOCYTES AND GIANT CELLS ALSO SEEN. NORMAL BRAIN PARENCHYMA INCLUDED IN SECTIONS | NA | NA | NA | NA | NA |
| 7 | 127683 | FASCICLES AND WHORLS. INDIVIDUAL TUMOR CELLS ARE ROUND TO OVAL, HAVING VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM, THERE IS EXTENSIVE HYALINIZATION AND DYSTROPHIC CALCIFICATION. PSOMMAMA BODIES ALSO IDENTIFIED. HOWEVER NO SHEETING PATTERN/NECROSIS/PROMINENT NUCLEOLI/SMALL CEL | NA | NA | NA | NA | NA |
| 8 | 130633 | SENCE OF A CELLULAR TUMOR WITH INTERSPERSED VARIABLY SIZED SMALL TO MEDIUM HYALINIZED BLOOD VESSELS. INDIVIDUAL TUMOR CELLS HAVE ROUND TO OVAL NUCLEI, FINE NUCLEAR CHROMATIN & MODERATE AMOUNT OF ILL DEFINED CYTOPLASM. OCCASIONAL BIZARRE FORMS IDENTIFIED. NO SHEETING /NECROSIS/MITOSIS /PROMINENT NUCLEOLI/SMAL | NA | NA | NA | NA | NA |
| 9 | 130973 | ELLS ARRANGED IN LOBULES, NESTS AND WHORLS. INDIVIDUAL TUMOR CELLS HAVE ROUND VESICULAR NUCLEI AND MODERATE AMOUNT OF ILL DEFINED EOSINOPHILIC CYTOPLASM. OCCASIONAL INTRANUCLEAR CYTOPLASMIC INCLUSIONS AND PSAMOMMA BODIES NOTED. NO SHEETING/SIGNIFICANT MITOSIS/NUCLEAR ATYPIA/SMALL CELLS SEEN. NO NORMAL BRA | NA | NA | NA | NA | NA |
| 10 | 133363 | TIONS SHOW PRESENCE OF A CELLULAR TUMOR ARRANGED IN THE HYPERCELLULAR AREAS (ANTONI A) AND HYPOCELLULAR AREAS (ANTONI B). THE ANTONI A AREAS SHOW PRESENCE OF SPINDLE WAVY NUCLEI WITH ILL DEFINED CYTOPLASM ARRANGED IN PALLISADED FASHION WITH VEROCAY BODIES. THERE IS MILD NUCLEAR HYPERCHROMASIA & PLEOMORPH | NA | NA | NA | NA | NA |
| 11 | 141564 | LOVULES AND NESTS OF MENINGOTHELIAL CELLS FOCALLY ARRANGED IN SYNCYTIAL PATTERN. AREAS OF CYSTIC CHANGE NOTED ALONG WITH INDIVIDUAL TUMOR CELLS EXHIBITING CYTOPLASMIC VACUOLATION. NUMEROUS BLOOD VESSELS ALONG WITH HYALINIZED STROMA ALSO EVIDENT. FEW OF THE TUMOR CELLS EXHIBIT PLEOMORPHIC NUCLEI HOWEVER, | NA | NA | NA | NA | NA |
| 12 | 138768 | S AND SYNCITIAL ARRANGEMENT.INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI AND ILL DEFINED CYTOPLASMIC BOUNDARIES. NUMEROUS BLOOD VESSELS WITH THICK HYALINISED WALLS NOTED.FEW PSAMMOMA BODIES IDENTIFIED. TINY BIT OF BRAIN PARENCHYMA INCLUDED DOES NOT SHOW TUMOR INFILTRATION. NO SMALL ( | NA | NA | NA | NA | NA |
| 13 | 143469 | SENCE OF A CELLULAR TUMOR WITH INDIVIDUAL TUMOR CELLS EXHIBITING NUCLEAR PLEOMORPHISM AND HYPERCHROMASIA PRESENT AGAINST A FIBRILLARY BACKGROUND.MITOTIC FIGURES NOTED. TINY FOCI OF NECROSIS PALISADED BY TUMOR CELLS ALSO NOTED.TUMOR CELLS ARE INFILTRATING INTO ADJACENT BRAIN PRENCHYMA. NO ENDOTHELIAL CELL PR | NA | NA | NA | NA | NA |
| 14 | 143900 | HOW A CELLULAR TUMOR WITH TUMOR CELLS EXHIBITING MINIMAL PLEOMORPHISM, HAVING ROUND NUCLEI, INDISTINCT CYTOPLASMIC BOUNDARIES ARRANGED AROUND A FIBRILLARY BACKGROUND.MICROCYSTS NOTED AND FEW GEMISTIOCYTES IDENTIFIED. OCCASIONAL MITOTIC FIGURES SEEN. HOWEVER NO NECROSIS/ SIGNIFICANT CYTOLOGICAL ATYPIA/ VAS | NA | NA | NA | NA | NA |
| 15 | 144018 | M THE BRAIN SHOWS A TUMOR WITH EXTENSIVE NECROSIS. TUMOR CELLS ARE ARRANGED IN SHEETS AND ARE PRESENT AGAINST A FIBRILLARY BACKGROUND. THE INDIVIDUAL TUMOR CELLS EXHIBIT MODERATE NUCLEAR PLEOMORPHISM AND HYPERCHROMASIA .ENDOTHELIAL PROLIFERATION ALSO NOTED ALONG WITH PSEUDOPALISADING AROUND THE AREAS. | NA | NA | NA | NA | NA |
| 16 | 149356 | TIONS SHOW PRESENCE OF A TUMOR WITH TUMOR CELLS ARRANGED IN LOBULES SURROUNDED BY FIBROCOLLAGENOUS SEPTAE. INDIVIDUAL TUMOR CELLS HAVE OVAL NUCLEI WITH INCONSPICUOUS NUCLEOLI. FOCAL WHORLING PATTERN AND PSAMMOMA BODIES IDENTIFIED .NO SHEETING PATTERN / ATYPICAL MITOSIS / NECROSIS SEEN IN THE SECTIONS EXAMI | NA | NA | NA | NA | NA |
| 17 | 16896 | SECTIONS FROM BRAIN SHOW LARGE AREAS OF NECROSIS, WITHIN WHICH ARE SEEN NESTS OF TUMOR CELLS HAVING VESICULAR NUCLEI, PROMINENT NUCLEOLI AND ABUNDANT AMOUNT OF CLEAR TO EOSINOPHILIC CYTOPLASM. CHOLESTEROL CLEFTS IDENTIFIED. MANY DILATED AND CONGESTED BLOOD VESSELS SEEN. | NA | NA | NA | NA | NA |
| 18 | 125404 | WITH TUMOR CELLS ARRANGED IN WHORLS AND SYNCITIUM. THE INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM WITH INDISTINCT CYTOPLASMIC BOUNDARIES. FOCI OF NECROSIS SEEN. AN OCCASIONAL MITOTIC FIGURE SEEN. TINY BIT OF NORMAL BRAIN PARENCHYMA INCLUDED IN SHO | NA | NA | NA | NA | NA |
| 19 | 136787 | ENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN DIFFUSE SHEETS AND PALISADED AROUND VASCULAR CHANNELS FORMING PSEUDOROSETTES. TRUE ROSETTE ALSO IDENTIFIED. TUMOR CELLS HAVE ROUND NUCLEI WITH MODERATE CYTOPLASM AND FIBRILLARY BACKGROUND. SMALL FOCI OF NECROSIS SEEN. TINY BIT OF NORMAL BRAIN PARENCH | NA | NA | NA | NA | NA |
| 20 | 145145 | THE INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM HAVING INDISTINCT CYTOPLASMIC BOUNDARIES. INTRANUCLEAR CYTOPLASMIC INCLUSION SEEN. FEW PSAMMOMA BODIES ALSO IDENTIFIED. TINY BIT OF NORMAL BRAIN PARENCHYMA SEEN DOES NOT SHOW INFILTRATION BY TUMOR CELLS. N | NA | NA | NA | NA | NA |
| 21 | 147199 | IBIT FOCAL PLEOMORPHISM WITH VESICULAR NUCLEI, PROMINENT NUCLEOLI, MODERATE AMOUNT OF CYTOPLASM WITH INDISTINCT CYTOPLASMIC BOUNDARIES.NUMEROUS DILATED AND CONGESTED BLOOD VESSELS IDENTIFIED. ALSO SEEN ARE OCCASIONAL PSAMMOMA BODIES. TINY AMOUNT OF NORMAL BRAIN PARENCHYMA ALSO SEEN, WHICH IS NOT INFILTRA | NA | NA | NA | NA | NA |
| 22 | 111671 | INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI, MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CYTOPLASMIC BOUNDARIES. FEW PSAMMOMA BODIES IDENTIFIED. ALSO SEEN ARE FEW HYALINISED AND CONGESTED BLOOD VESSELS. TINY BIT OF NORMAL BRAIN PARENCHYMA SEEN WHICH IS NOT INFILTRATED BY TUMOR CEL | NA | NA | NA | NA | NA |
| 23 | 152351 | WITH CELLS ARRANGED IN DIFFUSE SHEETS.TUMOR CELLS HAVE ROUND NUCLEI WITH ABUNDANT AMOUNT OF CYTOPLASM HAVING PERINUCELAR .MINIGEMISTIOCYTES ALSO IDENTIFIED. CYSTIC CHANGE ALSO SEEN. THE TUMOR CELLS ARE INFILTRATING INTO THE ADJACENT BRAIN PARENCHYMA. FOCI OF CALCIFICATION NOTED.HOWEVER, NO SIGNIFICANT/CYTO | NA | NA | NA | NA | NA |
| 24 | 154597 | G BIPHASIC PATTERN. INDIVIDUAL TUMOR CELLS HAVE MILDLY PLEOMORPHIC NUCLEI, PROMINENT NUCLEOLI AND ILL DEFINED CYTOPLASM AGAINST A FIBRILLARY BACKGROUND. PROMINENT MICROCYTIC PATTERN WITH ROSTHENTHAL FIBRES AND EOSINOPHILIC GRANULAR BODIES SEEN. AREAS OF HAEMORRHAGE AND FOCI OF PSEUDO-OLIGODENDROCYTE SEEN. | NA | NA | NA | NA | NA |
| 25 | 157839 | LAR TUMOR WITH TUMOR CELLS ARRANGED IN DIFFUSE SHEETS AGAINST A FIBRILLARY BACKGROUND. THE CELLS EXHIBIT NUCLEAR PLEOMORPHISM AND ANDHYPERCHROMASIA. THE TUMOR CELLS ARE INFILTRATING INTO ADJACENT BRAIN PARENCHYMA. PROMINENT CYSTIC CHANGE NOTED. HOWEVER, NO SIGNIFICANT MITOSIS/ENDOTHELIAL CELL PROLIFERAT | NA | NA | NA | NA | NA |
| 26 | 158146 | UMOR WITH INCREASED CELLULARITY.INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC AND HYPERCHROMATIC NUCLEI WHICH ARE PRESENT AGAINST A FIBRILLARY BACKGROUND, FEW MITOTIC FIGURES SEEN WITH AREAS OF NECROSIS PALISADED BY THE TUMOR CELLS. AREAS OF HAEMORRHAGE IDENTIFIED. WITHIN AREAS OF NECROSIS BLOOD VESSELS WITH TH | NA | NA | NA | NA | NA |
| 27 | 163971 | . MAJORITY OF THE AREA SHOW MILD NUCLEAR PLEOMORPHISM AND HYPERCHROMASIA – GRADE II ASTROCYTOMA. FOCAL AREA SHOW INCREASED TUMOR CELLULARITY ALONG WITH MITOSIS AND AN OCCASIONAL GIANT CELL. TUMOR CELLS SHOW FOCAL PALISADED ARRANGEMENT AROUND THE BLOOD VESSELS. MANY GEMISTIOCYTES IDENTIFIED. TUMOR CELLS | NA | NA | NA | NA | NA |
| 28 | 173351 | SECTIONS TAKEN FROM THE BLOOD VESSEL SHOWS PRESENCE OF BLOOD CLOT MAINLY. GMS AND PAS STAIN FOR FUNGUS ARE NEGATIVE. | NA | NEGATIVE | NEGATIVE | NA | NA |
| 29 | 173888 | [redacted] | NA | NA | NA | NA | NA |
| 30 | 175212 | SECTIONS SHOW PRESENCE OF BLOOD CLOT MAINLY. NO NORMAL BRAIN PARENCHYMA/EVIDENCE OF MALIGNANCY SEEN IN SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 31 | 174863 | IONS FROM BRAIN SHOW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL CELLS HAVE HYPERCHROMATIC, MILDLY PLEOMORPHIC NUCLEI. PROMINENT CYSTIC CHANGE & FEW GEMISTIOCYTES NOTED. HOWEVER, NO NECROSIS/ENDOTHELIAL CELL PROLIFERATION/SIGNIFICANT MITOSIS IDENT | NA | NA | NA | NA | NA |
| 32 | 177170 | NGED IN SHEETS.INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEI WITH A PERINUCLEAR HALO & WELL DEFINED CELL MEMBRANES. CHICKEN WIRE VASCULATURE WITH MICROVASCULAR PROLIFERATION SEEN. MITOTIC FIGURES WITH AREAS OF CALCIFICATION NOTED.THE TUMOR CELLS ARE INFILTRATING INTO THE ADJACENT NORMAL BI | NA | NA | NA | NA | NA |
| 33 | 178484 | N FROM BOTH BIOPSIES SHOW PRESENCE OF NUMEROUS EPITHELIOID CELL GRANULOMAS WITH LANGHANS AND FOREIGN BODY GIANT CELL REACTION ALONG WITH PRESENCE OF AREAS OF NECROSIS. DENSE NEUTROPHILIC INFILTRATE ALSO IDENTIFIED AT PLACES.HOWEVER, NO FUNGAL HYPHAE/SPORES/EVIDENCE OF MALIGNANCY SEEN IN THE MULTIPLE SECT | NEGATIVE | NEGATIVE | NEGATIVE | NA | NA |
| 34 | 175983 | HISTOMORPHOLOGICAL FEATURES ARE CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 35 | 179096 | ULAR TUMOR WITH TUMOR CELLS ARRANGED IN CLUSTERS WITH TUBULES AND INDIAN FILE PATTERN. INDIVIDUAL TUMOR CELLS HAVE MODERATELY PLEOMORPHIC VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF EOSINOPHILIC CYTOPLASM WITH CYTOPLASMIC VACUOLES. EXTENSIVE AREAS OF NECROSIS IDENTIFIED. INFILTRATION UPTO T | NA | NA | NA | NA | NA |
| 36 | 183188 | ELLS ARRANGED IN WHORLS AND FASCICLES. INDIVIDUAL TUMOR CELLS ARE OVAL HAVING BLAND NUCLEI AND ILL DEFINED CYTOPLASM. LARGE AREAS OF NECROSIS WITH ACUTE INFLAMMATORY INFILTRATE SEEN. ALSO SEEN ARE SCATTERED PSAMMOMA BODIES. AN OCCASIONAL MITOTIC FIGURES IDENTIFIED.HOWEVER, NO SHEETING/INCREASE CELLULARITY/SM | NA | NA | NA | NA | NA |
| 37 | 167340 | SECTIONS FROM BRAIN SHOW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN INTERSECTING FASCICLES,WHORLS AND LOBULES. INDIVIDUAL TUMOR CELLS HAVE ROUND TO OVAL VESICULAR NUCLEI ,INCOSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CYTOPLASMIC BOUNDARIES. | NA | NA | NA | NA | NA |
| 38 | 184118 | HISTOMORPHOLOGICAL FEATURES ARE CONSISTENT WITH PITUITARY ADENOMA WITH AREAS OF NECROSIS. | NA | NA | NA | NA | NA |
| 39 | 184700 | ULAR AREAS. TUMOR CELLS HAVE ELONGATED WAVY NUCLEI AND MODERATE AMOUNT OF EOSINOPHILIC CYTOPLASM. NUCLEAR PLEOMORPHISM & HYPERCHROMASIA ALSO NOTED. MANY VEROCAY BODIES IDENTIFIED. HYPOCELLULAR AREAS SHOW CYSTIC CHANGE WITH FEW THICKENED & HYALINISED BLOOD VESSELS AND MILD CHRONIC PERIVASCULAR INFLA | NA | NA | NA | NA | NA |
| 40 | 187900 | HISTOLOGY CONSISTENT WITH BLOOD CLOT. | NA | NA | NA | NA | NA |
| 41 | 191139 | A CELLULAR TUMOR COMPOSED OF ALTERNATING HYPERCELLULAR AND HYPOCELLULAR AREAS.HYPERCELLULAR AREAS CONSIST OF CELLS HAVING BUCKLE SHAPED NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. HYPOCELLULAR AREAS SHOW CYSTIC CHANGE WITH FEW THICKENED & HYALINISED BLOOD VESSELS AND MILD CHRONIC PERIVASCULAR INFLA | NA | NA | NA | NA | NA |
| 42 | 1696 | D VESSELS LINED BY ENDOTHELIUM. IN BETWEEN ARE SEEN TUMOR CELLS IN NESTS WITH INDIVIDUAL TUMOR CELLS HAVING ROUND TO OVAL VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF EOSINOPHILIC CYTOPLASM. MILD NUCLEAR PLEOMORPHISM AND HYPERCHROMASIA NOTED.HOWEVER, NO SHEETING ARRANGEMENT NECROSIS/ | NA | NA | NA | NA | NA |
| 43 | 193975 | PRESENCE OF CELLULAR TUMOR WITH CELLS ARRANGED IN DIFFUSE SHEETS AND FOCALLY PALISADED AROUND AREAS OF NECROSIS. THE TUMOR CELLS EXHIBIT MODERATE, PLEOMORPHISM, HYPERCHROMASIA AND ARE PRESENT AGAINST FIBRIALLY BACKGROUND. MITOTIC FIGURES, AREAS OF NECROSIS AND ENDOTHELIAL PROLIFERATION FORMING GLOMERUI | NA | NA | NA | NA | NA |
| 44 | 198641 | BRAIN SHOW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN NESTS SEPARATED BY THIN FIBROVASCULAR SEPTAE. INDIVIDUAL TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI WITH ABUNDANT AMOUNT OF CLEAR CYTOPLASM AND WELL DEFINED CYTOPLASMIC BOUNDARIES. LARGE AREA OF HAEMORRHAGE NOTED WITH HEMOSIDERIN LA | NA | NA | NA | NA | NA |
| 45 | 197185 | ULAR TUMOR WITH CELLS ARRANGED IN SHEETS AND HAVE HYPERCHROMATIC NUCLEI EXHIBITING MILD NUCLEAR PLEOMORPHISM. FOCAL CHICKEN WIRE VASCULATURE NOTED IN THE BACKGROUND.TUMOR IS INFILTRATING THE ADJACENT BRAIN PARENCHYMA WITH PERINEURONAL SATELLITOSIS. CYSTIC CHANGE SEEN. NO SIGNIFICANT MITOTIS/NECROSIS/ENDO | NA | NA | NA | NA | NA |
| 46 | 187539 | F A CELLULAR TUMOR. THE TUMOR CELLS ARE ARRANGED IN DIFFUSE SHEETS AND INFILTRATING ADJACENT NORMAL BRAIN PARENCHYMA. THE TUMOR CELLS HAVE MILDLY PLEOMORPHIC HYPERCHROMATIC NUCLEI PRESENT AGAINST A FIBRILLARY BACKGROUND.MANY GEMISTOCYTES IDENTIFIED. MITOTIC FIGURES SEEN. AREAS OF NECROSIS AND ENDOTHELIAL P | NA | NA | NA | NA | NA |
| 47 | 202394 | H CELLS ARRANGED IN WHORLS, SHORT FASCICLES AND OCCASIONAL SHEETS. THEY ARE SEPARATED BY DENSE COLLAGEN BUNDLES. THE NUCLEI ARE OVAL TO SPINDLE SHAPED WITH INDISTINCT NUCLEOLI. MILD NUCLEAR PLEOMORPHISM, OCCASIONAL MITOTIC FIGURES < 4/10 HPF, AND EXTENSIVE AREAS OF NECROSIS IDENTIFIED.FEW PSAMMOMA BODIES & M | NA | NA | NA | NA | NA |
| 48 | 202931 | SECTIONS SHOW PRESENCE OF A TUMOR WITH TUMOR CELLS ARRANGED IN LOBULES AND WHORLS WITH INDIVIDUAL TUMOR CELLS HAVING OVAL NUCLEI WITH MODERATE AMOUNT OF CYTOPLASM. MANY PSAMMOMA BODIES IDENTIFIED. HOWEVER, NO CYTOLOGICAL ATYPIA/MITOTIC FIGURES IDENTIFIED IN THE MULTIPLE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 49 | 207921 | LS ARRANGED IN HYPOCELLULAR & HYPERCELLULAR AREAS. HYPERCELLULAR AREAS ARE SEEN AS CELLS HAVING ELONGATED VESICULAR NUCLEI ILL DEFINED EOSINOPHILIC CYTOPLASM WITH VEROCAY BODIES. HYPOCELLULAR AREAS SHOW PRESENCE OF SPINDLE SHAPED CELLS WITH VESICULAR NUCLEI & MODERATE AMOUNT OF CYTOPLASM ALONG WITHY C | NA | NA | NA | NA | NA |
| 50 | 206237 | SECTIONS SHOW A CELLULAR TUMOR AGAINST A FIBRILLARY BACKGROUND.THE CELLS EXHIBIT MODERATE CELLULAR PLEOMORPHISM AND OCCASIONAL MITOSIS.NUMEROUS GEMISTOCYTES PRESENT. THE TUMOR IS SEEN INFILTRATING BRAIN PARENCHYMA & REACHING UPTO SUBPIAL LOCATION. CYSTIC CHANGE ALSO SEEN | NA | NA | NA | NA | NA |
| 51 | 208396 | ENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. MOST OF TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI AND MANY CONTAIN PERINUCLEAR HALO- OLIODENDROGLIAL COMPONENT. MICROGEMISTOCYTES SEEN. CHICKEN WIRE VASCULATURE WITH ENDOTHELIAL PROLIFERATION, MITOTIC FIGURES A | NA | NA | NA | NA | NA |
| 52 | 61337 | IN WHORLS AND FASCICLES.INDIVIDUAL TUMOR CELLS HAVE ROUND TO OVAL NUCLEI EXHIBITING MILD NUCLEAR PLEOMORPHISM WITH NUCLEAR CLEARING AT PLACES. FOCI OF NECROSIS AND OCCASIONAL MITOTIC FIGURES IDENTIFIED. HOWEVER, NO SHEETING PATTERN/SIGNIFICANT MITOSIS/SMALL CELLS SEEN IN THE SECTIONS EXAMINED. TINY BIT OF BRAIN P | NA | NA | NA | NA | NA |
| 53 | 86271 | SECTIONS FROM THE BRAIN SHOW CELLULAR TUMOR ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS ARE MEDIUM TO LARGE SIZED HAVING VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. BRISK MITOSIS WITH MANY APOPTOTIC BODIES IDENTIFIED.FOCI OF NECROSIS ALSO SEEN. | NA | NA | NA | NA | NA |
| 54 | 197175 | AKEN FROM THE BRAIN SHOW DENSE INFILTRATION OF THE PARENCHYMA BY ACUTE AND CHRONIC INFLAMMATORY CELLS WITH FOCI OF NECROSIS. MANY DILATED AND CONGESTED BLOOD VESSELS ALSO SEEN. WITHIN THE NECROTIC AREAS ARE SEEN FEW SEPTATE HYPHAE OF ASPERGILLUS. NO GRANULOMA/EVIDENCE OF MALIGNANCY SEEN IN THE SECTIONS | NA | POSITIVE | POSITIVE | NA | NA |
| 55 | 34673 | SECTIONS TAKEN FROM THE BRAIN SHOW PRESENCE OF CELLULAR TUMOR INFILTRATING THE BRAIN PARENCHYMA WITH CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. BRISK MITOSIS SEEN. | NA | NA | NA | NA | NA |
| 56 | 181419 | SECTION FROM SOFT TISSUE AND BONE IN BOTH THE CONTAINERS SHOW PRESENCE OF A TUMOR WITH CELLS ARRANGED IN SHEETS AND NESTS. THE TUMOR CELLS ARE LARGE AND HAVE ROUND VESICULAR NUCLEI, PROMINENT NUCLEOLI & MODERATE AMOUNT OF CYTOPLASM. BRISK MITOSIS SEEN ALONG WITH AREAS OF NECROSIS & HAEMORRHAGE | NA | NA | NA | NA | NA |
| 57 | 214430 | A CELLULAR TUMOR COMPOSED OF ALTERNATING HYPERCELLULAR AND HYPOCELLULAR AREAS.HYPERCELLULAR AREAS CONSIST OF CELLS HAVING BUCKLE SHAPED NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. HYPOCELLULAR AREAS SHOW PALISADING OF NUCLEI-VERROCAY BODIES WITH MYXOID CHANGE. HOWEVER, NO SIGNIFICANT MITOSIS/NECROSIS S | NA | NA | NA | NA | NA |
| 58 | 210811 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA- SPACE OCCUPYING LESION BRAIN. | NA | NA | NA | NA | NA |
| 59 | 142918 | S SHOW PRESENCE OF A CELLULAR TUMOR ARRANGED IN WHORLS AND FASCICLES.INDIVIDUAL TUMOR CELLS ARE ROUND TO OVAL TO SPINDLE SHAPED HAVING VESICULAR NUCLEI, INCONSPICOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM.PSAMMOMA BODIES SEEN. HOWEVER, NO NORMAL BRAIN PARENCHYMA IDENTIFIED IN THE MULTIPLE SECTIONS E | NA | NA | NA | NA | NA |
| 60 | 219156 | OR WITH TUMOR CELLS ARRANGED IN SHEETS.INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM AGAINST A FIBRILLARY BACKGROUND. MITOTIC FIGURES AND TUMOR GIANT CELLS ALSO IDENTIFIED.AREAS OF EXTENSIVE NECROSIS WITH TUMOR CELLS PALISADED AROUND OCCASIC | NA | NA | NA | NA | NA |
| 61 | 219841 | HISTOLOGY CONSISTENT WITH BLOOD CLOT. | NA | NA | NA | NA | NA |
| 62 | 221657 | MOR CELLS ARRANGED IN WHORLS & FASCICLES. THE INDIVIDUAL TUMOR CELLS HAVE ROUND VESICULAR NUCLEI AND MODERATE CYTOPLASM HAVING ILL DEFINED CYTOPLASMIC BOUNDARIES. PSAMMOMA BODIES SEEN. NORMAL BRAIN PARENCHYMA ALSO IDENTIFIED WHICH IS NOT INFILTRATED BY TUMOR CELLS. FOCI OF NECROSIS SEEN.NO SHEETING/SMAL | NA | NA | NA | NA | NA |
| 63 | 203073 | ELLULAR TUMOR WITH PRESENCE OF TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND VESICULAR NUCLEUS. PERINEURONAL SATELLOTASIS NOTED. CHICKEN WIRE PATTERN OF BRANCHING CAPILLARIES IDENTIFIED.CYSTIC CHANGE ALSO SEEN. NO NECROSIS/ENDOTHELIAL CELL PROLIFERATION/SIGNIFICANT NUCLEAR PLEOMORPHIS | NA | NA | NA | NA | NA |
| 64 | 221526 | FASCICLES AND WHORLS. THE INDIVIDUAL TUMOR CELLS HAVE OVAL VESICULAR NUCLEI AND HAVING ILL DEFINED CYTOPLASMIC BOUNDARIES. FOCI OF NECROSIS WITH AREAS OF CALCIFICATION SEEN. LYMPHOCYTIC INFILTRATE WITH FOAM CELL CHANGE NOTED. NORMAL BRAIN PARENCHYMA ALSO IDENTIFIED WHICH IS NOT INFILTRATED BY TUMOR CELLS. NO SH | NA | NA | NA | NA | NA |
| 65 | 221014 | ENCE OF NORMAL LOOKING BRAIN PARENCHYMA WITH ONE FOCAL AREA SHOWING A CELLULAR TUMOR WITH INCREASE CELLULARITY & PROMINENT CYSTIC CHANGE.THE TUMOR CELLS HAVE ROUND NUCLEI WITH PRESENCE OF ? FIBRILLARY BACKGROUND.HOWEVER, NO AREA OF NECROSIS/SIGNIFICANT MITOSIS/ENDOTHELIAL CELL PROLIFERATION IDENTIFIED IN I | NA | NA | NA | NA | NA |
| 66 | 128564 | CE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN FASCICLES AND WHORLS. INDIVIDUAL TUMOR CELLS HAVE OVAL TO SPINDLE SHAPED VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. PSAMMOMA BODIES IDENTIFIED. NO NORMAL BRAIN PARENCHYMA SEEN.NO SHEETING /MITOSIS/NECROSIS/SMALL CELL CHANGE SEEN IN | NA | NA | NA | NA | NA |
| 67 | 231442 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 68 | 203389 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 69 | 23086 | S SHOW PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN DIFFUSE SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND ENLARGED NUCLEI WITH ABUNDANT AMOUNT OF CYTOPLASM & PERINUCLEAR HALO. PERINEURAL SATELLOTOSIS NOTED. CHICKEN WIRE CALCIFICATION IDENTIFIED. NO SIGNIFICANT NECROSIS/MITOSIS NOTED IN THE SECTIONS E | NA | NA | NA | NA | NA |
| 70 | 240675 | SECTIONS SHOW CELLULAR TUMOR WITH CELLS ARRANGED INWHORLS AND FASCICLES. INDIVIDUAL TUMOR CELLS ARE OVAL TO ELONGATED WITH VESICULAR NUCLEI AND ILL DEFINED CYTOPLASMIC BOUNDARIES. AREAS OF NECROSIS AND AN OCCASIONAL MITOTIC FIGURE IDENTIFIED. NO NORMAL BRAIN PARENCHYMA SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 71 | 240753 | CAPSULATED TUMOR COMPRISING OF HYPOCELLULAR AND HYPERCELLULAR AREAS. HYPOCELLULAR AREAS SHOW PROMINENT MYXOID CHANGE AND CYSTIC CHANGE. HYPERCELLULAR AREA IS COMPOSED OF SPINDLE SHAPED CELLS HAVING BUCKLED NUCLEI AND ILL DEFINED CYTOPLASMIC BOUNDARIES. FEW VEROCAY BODIES IDENTIFIED. MANY THICK WALLED | NA | NA | NA | NA | NA |
| 72 | 242845 | NECROSIS.THE TUMOR CELLS ARE ARRANGED IN DIFFUSE SHEETS AS WELL AS PALISADED AROUND AREAS OF NECROSIS & BLOOD VESSELS. THE TUMOR CELLS EXHIBIT MARKED NUCLEAR PLEOMORPHISM HAVING HYPERCHROMATIC NUCLEI AND PRESENT AGAINST FIBRILLARY BACKGROUND. FEW MITOTIC FIGURES ALONG WITH TUMOR GIANT CELLS IDENTIFIED. N | NA | NA | NA | NA | NA |
| 73 | 247484 | MULTIPLE SECTIONS TAKEN SHOW BRAIN PARENCHYMA HAVING AREAS OF INTRAPARENCHYAL HAEMORRHAGE & FOCAL CYSTIC CHANGE. FEW DILATED & CONGESTED BLOOD VESSELS ALS

| SR. NO. | MRD | CONGO RED | IMMUNO-HISTOCHEMISTRY | | | | | | | | | FINAL HISTOPATHOLOGICAL DIAGNOSIS | GRADE | IDH STATUS | CATEGORY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | GFAP | ATRX | IDH-1 | MIB-1 | P53 | CK | EMA | S100 | OTHER | | | | |
| 1 | 67353 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANGIOMATOUS MENINGIOMA | I | NA | NEOPLASTIC |
| 2 | 77522 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC OLIGOASTROCYTOMA | III | NA | NEOPLASTIC |
| 3 | 83867 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MEDULLOBLASTOMA | IV | NA | NEOPLASTIC |
| 4 | 118798 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 5 | 122689 | NA | NA | NA | NA | 16% | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 6 | 111621 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 7 | 127683 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 8 | 130633 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANGIOMATOUS MENINGIOMA | I | NA | NEOPLASTIC |
| 9 | 130973 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 10 | 133363 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 11 | 141564 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MICROCYSTIC MENINGIOMA | I | NA | NEOPLASTIC |
| 12 | 138768 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 13 | 143469 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 14 | 143900 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 15 | 144018 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 16 | 149356 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | I | NA | NEOPLASTIC |
| 17 | 16896 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 18 | 125404 | NA | NA | NA | NA | 8% | NA | NEGATIVE | POSITIVE | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 19 | 136787 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPENDYMOMA | II | NA | NEOPLASTIC |
| 20 | 145145 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 21 | 147199 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANGIOMATOUS MENINGIOMA | I | NA | NEOPLASTIC |
| 22 | 111671 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 23 | 152351 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 24 | 154597 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PILOCYTIC ASTROCYTOMA | I | NA | NEOPLASTIC |
| 25 | 157839 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 26 | 158146 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 27 | 163971 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC ASTROCYTOMA | III | NA | NEOPLASTIC |
| 28 | 173351 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANEURYSM | NA | NA | NON-NEOPLASTIC |
| 29 | 173888 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ABSCESS | NA | NA | NON-NEOPLASTIC |
| 30 | 175212 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 31 | 174863 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 32 | 177170 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC OLIGODENDROGLIOMA | III | NA | NEOPLASTIC |
| 33 | 178484 | NA | NA | NA | NA | NA | NA | NA | NA | NA | - | GRANULOMATOUS INFLAMMATION -? TUBERCULOSIS | NA | NA | NON-NEOPLASTIC |
| 34 | 175983 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 35 | 179096 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 36 | 183188 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC MENINGIOMA | III | NA | NEOPLASTIC |
| 37 | 167340 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 38 | 184118 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 39 | 184700 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 40 | 187900 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 41 | 191139 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 42 | 1696 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANGIOMATOUS MENINGIOMA | I | NA | NEOPLASTIC |
| 43 | 193975 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 44 | 198641 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 45 | 197185 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 46 | 187539 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 47 | 202394 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ATYPICAL MENINGIOMA | II | NA | NEOPLASTIC |
| 48 | 202931 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PSAMMOMATOUS MENINGIOMA | I | NA | NEOPLASTIC |
| 49 | 207921 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 50 | 206237 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC ASTROCYTOMA | III | NA | NEOPLASTIC |
| 51 | 208396 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC OLIGODENDROGLIOMA | III | NA | NEOPLASTIC |
| 52 | 61337 | NA | NA | NA | NA | 25% | NA | NA | NA | NA | EMA POSITIVE | ANAPLASTIC MENINGIOMA | I | NA | NEOPLASTIC |
| 53 | 86271 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | NON HODGKIN LYMPHOMA - DLBCL | NA | NA | NEOPLASTIC |
| 54 | 197175 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ASPERGILLOSIS | NA | NA | NON-NEOPLASTIC |
| 55 | 34673 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 56 | 181419 | NA | NA | NA | NA | 70% | NA | POSITIVE | POSITIVE | NEGATIVE | CD10, VIMENTIN - POSITIVE & DESMIN, P63, HEP PAR-1, TTF-1, PSA, LCA, CK-7, CK-20, S-100, CD-30, CD-117 - NEGATIVE | METASTATIC CARCINOMATOUS DEPOSITS - LIKELY RENAL | NA | NA | NEOPLASTIC |
| 57 | 214430 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 58 | 210811 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 59 | 142918 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 60 | 219156 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 61 | 219841 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 62 | 221657 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 63 | 203073 | NA | NEGATIVE | NA | NA | 10% | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 64 | 221526 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | I | NA | NEOPLASTIC |
| 65 | 221014 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 66 | 128564 | NA | NA | NA | NA | - | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | II | NA | NEOPLASTIC |
| 67 | 231442 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 68 | 203389 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 69 | 23086 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 70 | 240675 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | II | NA | NEOPLASTIC |
| 71 | 240753 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 72 | 242845 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 73 | 247484 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | INTRAPARENCHYMAL HEMORRHAGE | NA | NA | NON-NEOPLASTIC |
| 74 | 260907 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 75 | 272283 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | AMOEBIC ABSCESS | NA | NA | NON-NEOPLASTIC |
| 76 | 278682 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 77 | 289978 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 78 | 293837 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | FIBROBLASTIC MENINGIOMA | I | NA | NEOPLASTIC |
| 79 | 320492 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 80 | 339045 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 81 | 341494 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 82 | 347512 | NA | NA | NEGATIVE | NEGATIVE | 10% | NA | NA | NA | NA | NA | ANAPLASTIC ASTROCYTOMA | III | WILD TYPE | NEOPLASTIC |
| 83 | 357669 | NA | POSITIVE | NA | NA | 50% | NA | NA | NA | NA | SYNAPTOPHYSIN - POSITIVE & LCA - NEGATIVE | MEDULLOBLASTOMA | IV | NA | NEOPLASTIC |
| 84 | 360383 | NA | NA | NA | NA | 90% | NA | NA | POSITIVE | NEGATIVE | CK7, WT1 - POSITIVE & CK20 - NEGATIVE | METASTATIC CARCINOMATOUS DEPOSITS - LIKELY CA OVARY | NA | NA | NEOPLASTIC |
| 85 | 373685 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 86 | 374039 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PACHYMENINGITIS | NA | NA | NON-NEOPLASTIC |
| 87 | 378279 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANGIOMATOUS MENINGIOMA | I | NA | NEOPLASTIC |
| 88 | 388390 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 89 | 391144 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDERMAL INCLUSION CYST | NA | NA | NON-NEOPLASTIC |
| 90 | 173351 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 91 | 396053 | NA | NA | NA | NA | 50% | NA | NA | NA | NA | LCA, CD20, CD3, MUM1,BCL2 - POSITIVE & TDT, BCL6,CD10, CD30 - NEGATIVE | NON HODGKIN LYMPHOMA - DLBCL | NA | NA | NEOPLASTIC |
| 92 | 398025 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 93 | 400904 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |

| SR. NO. | MRD | HISTO NO. | NAME | AGE | SEX | CHIEF COMPLAINT | PAST HISTORY | CLINICAL DIAGNOSIS | SITE OF BIOPSY | RADIOLOGY | GROSS EXAMINATION OF SPECIMEN |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 94 | 402201 | 14149-17 | HSK | 55 | M | HEADACHE | K/CO HYPERTENSION | TEMPORAL SOL | RIGHT TEMPORAL | INTRACRANIAL HAEMORRHAGE | MULTIPLE 5X3.5X1 CM |
| 95 | 401453 | 14228-17 | JS | 16 | M | HEADACHE & VOMITING | NA | TUMOR BRAIN TISSUE | SPHENOID SINUS | SHENOID SINUS SOL | MULTIPLE 5X3X2 CM |
| 96 | 402531 | 14251-17 | HS | 45 | M | HEADACHE,DIZZINESS & BLURING OF VISION | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 2X1.3X0.6 CM |
| 97 | 403644 | 14290-17 | AS | 22 | M | HEADACHE & WEAKNESS | NA | EPIDURAL TISSUE MASS | DURAL BIOPSY | NO SIGNIFICANT ABNORMALITY | MULTIPLE 7X4X2 CM |
| 98 | 401798 | 14292-17 | NK | 70 | F | HEADACHE & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 9X5X2 CM |
| 99 | 406646 | 14574-17 | GS | 42 | M | HEADACHE,DIZZINESS & BLURING OF VISION | NA | PITUITARY MACROADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 4X3X1 CM |
| 100 | 389792 | 14643-17 | KJ | 43 | F | HEADACHE,WEAKNESS & DIZZINESS | NA | PITUITARY MACROADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 1.5X1.1X0.5 CM |
| 101 | 412409 | 175-18 | KR | 50 | M | HEADACHE,ABSENSE OF SMELL & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL GLIOMA | RIGHT FRONTAL GLIOMA | HIGH GRADE GLIOMA | MULTIPLE 5X3X2 CM |
| 102 | 418434 | 310-18 | NS | 65 | M | HEADACHE,ABSENSE OF SMELL & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL SOL | FRONTAL INTRACRANIAL | METASTATIC CARCINOMA | SINGLE 4X3X1 CM |
| 103 | 417775 | 376-18 | PR | 41 | M | HEADACHE,DIZZINESS & SEIZURES | NA | BRAIN SOL | RIGHT TEMPORAL | MENINGIOMA | SINGLE 2.5X2X0.5 CM |
| 104 | 422241 | 640-18 | C | 60 | F | SEVERE HEADACHE,DIZZINESS & BLURING OF VISION | NA | ARTERIO-VENOUS MALFORMATION | LEFT TEMPORAL | ARTERIO VENOUS MALFORMATION | MULTIPLE 5X3X2 CM |
| 105 | 425016 | 955-18 | PK | 30 | F | HEADACHE,BEHAVIORAL CHANGES & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 5X2.5X0.4 CM |
| 106 | 431144 | 1287-18 | RK | 44 | M | HEADACHE,DIFFICULTY IN WALKING & LEFT SIDED WEAKNESS | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | LOW GRADE GLIOMA | MULTIPLE 8X6X3 CM |
| 107 | 431378 | 1367,1369 -18 | SS | 23 | M | LOSS OF SMELL & TASTE & FATIGUE | NA | MALIGNANT TUMOR | OLFACTORY GROOVE | LARGE HETEROGENOUS MASS | SINGLE 3X2X1 CM |
| 108 | 437630 | 1675-18 | KK | 50 | F | HEADACHE,DIZZINESS & SEIZURES | NA | MENINGIOMA | RIGHT FRONTO-TEMPORAL | MENINGIOMA | SINGLE 8X6X2 CM |
| 109 | 435876 | 1963-18 | BS | 42 | M | HEADACHE,WEAKNESS & DIZZINESS | NA | PITUITARY TUMOR | EXCISE PITUTARY MASS | ADENOMA OF PITUITARY GLAND | MULTIPLE 2X1X0.5 CM |
| 110 | 457525 | 2806-18 | NK | 50 | F | HEADACHE,VOMITING & WEAKNESS | NA | INTRACRANIL HAEMORRHAGE | RIGHT TEMPORO-PARIETAL | INTRACRANIL HAEMORRHAGE | MULTIPLE 4X3.5X1.5 CM |
| 111 | 460964 | 3099-18 | HK | 75 | F | HEADACHE,DIZZINESS & WEAKNESS | NA | LEFT FRONTAL NON HODGKINS LYMPHOMA | LEFT FRONTAL | LYMPHOMA | MULTIPLE 2.5X2X0.4 CM |
| 112 | 472333 | 3822-18 | GK | 70 | F | HEADACHE & ABSENSE OF SMELL | NA | LEFT FRONTAL SOL | LEFT FRONTAL | METASTATIC CARCINOMA | MULTIPLE 3.5X2X1 CM |
| 113 | 478141 | 4313-18 | KL | 40 | F | HEADACHE,DIZZINESS & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 1.5X1X0.5 CM |
| 114 | 485287 | 4922-18 | JS | 45 | M | HEADACHE,VOMITING & WEAKNESS IN LIMBS | NA | RIGHT FRONTO-TEMPORAL SOL ? MENINGIOMA | RIGHT FRONTO-TEMPORAL | MENINGIOMA | MULTIPLE 10X8X1 CM |
| 115 | 489703 | 5050-18 | HK | 72 | F | HEADACHE,VOMITING & WEAKNESS IN LIMBS | NA | MENINGIOMA | LEFT PARIETAL | MENINGIOMA | MULTIPLE 10X6X3 CM |
| 116 | 492864 | 5449-18 | SK | 50 | F | HEADACHE& WEAKNESS OF MUSCLES | NA | GLIOBLASTOMA MULTIFORME | RIGHT PARIETO-OCCIPITAL | HIGH GRADE GLIOMA | MULTIPLE 4X2X1 CM |
| 117 | 496018 | 5522-18 | U | 50 | F | HEADACHE & VOMITING | H/O HEAD TRAUMA | INTRACRANIL HAEMORRHAGE | LEFT TEMPORAL | INTRACRANIL HAEMORRHAGE | MULTIPLE 3X2X1 CM |
| 118 | 496293 | 5622-18 | PK | 12 | F | HEADACHE & BLURING OF VISION | NA | RIGHT TEMPORO-PARIETAL SOL | RIGHT TEMPORO-PARIETAL | LOW GRADE GLIOMA | MULTIPLE 2.5X2X1.5 CM |
| 119 | 464221 | 5738-18 | GS | 64 | M | HEADACHE,WEAKNESS & BLURING OF VISION | NA | ASTROCYTOMA | LEFT TEMPORO-PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 5X2.5X1.8 CM |
| 120 | 444920 | 5805-18 | UD | 36 | F | HEADACHE,VOMITING & WEAKNESS IN LIMBS | NA | LEFT FRONTAL TUMOR | LEFT FRONTAL TUMOR | MENINGIOMA | SINGLE 5X4X1 CM |
| 121 | 502444 | 6037-18 | BD | 52 | F | HEADACHE,VOMITING & MUSCLE WEAKNESS | NA | FRONTAL SOL | RIGHT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 122 | 505064 | 6563-18 | BK | 60 | F | HEADACHE,DECREASED VISION & WEAKNESS OF MUSCLES | NA | RIGHT PARIETAL BONE SOL | RIGHT PARIETAL BONE | METASTATIC CARCINOMA | MULTIPLE 4X3X1.5 CM |
| 123 | 516719 | 7139-18 | SD | 52 | F | HEADACHE & DIZZINESS | NA | LEFT TEMPOROPARIETAL SOL | LEFT TEMPORO-PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 2X1X0.5 CM |
| 124 | 532310 | 8191-18 | GK | 44 | F | HEADACHE & LEFT SIDED WEAKNESS | NA | INTRACRANIL HAEMORRHAGE | RIGHT PARIETO-OCCIPITAL | INTRACRANIAL HAEMORRHAGE | MULTIPLE 2X1.5X1 CM |
| 125 | 531353 | 8256-18 | KK | 50 | F | HEADACHE & BLURING OF VISION | NA | MENINGIOMA | LEFT PARIETAL | MENINGIOMA | MULTIPLE 6.5X5X1.3 CM |
| 126 | 532909 | 8469-18 | AK | 64 | F | HEADACHE,VOMITING & BLURING OF VISION | NA | MENINGIOMA | RIGHT FRONTO-PARIETAL | MENINGIOMA | MULTIPLE 4.4X5X1.2 CM |
| 127 | 531502 | 8475-18 | BR | 52 | M | HEADACHE & LEFT SIDED WEAKNESS | NA | RIGHT TEMPORAL MASS | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 6X5X4 CM |
| 128 | 541231 | 8848-18 | SK | 75 | F | HEADACHE,BLURING OF VISION & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL REGION SOL | LEFT FRONTAL | MENINGIOMA | MULTIPLE 10X6X3 CM |
| 129 | 542975 | 9224-18 | LD | 57 | F | HEADACHE & VOMITING | H/O HEAD TRAUMA | INTRACRANIAL HEMORRHAGE | RIGHT PARIETAL | THROMBUS FORMATION | MULTIPLE 4X3X2.5 CM |
| 130 | 542798 | 9404-18 | HS | 53 | M | HEADACHE,BLURING OF VISION & SEIZURES | NA | MENINGIOMA | LEFT PARIETAL AREA | HIGH GRADE GLIOMA | MULTIPLE 8X5X2 CM |
| 131 | 545674 | 9454-18 | GS | 54 | M | HEADACHE & BLURING OF VISION | NA | RIGHT FRONTAL REGION SOL | RIGHT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 7X6X1.5 CM |
| 132 | 506598 | 10194-18 | ND | 55 | F | HEADACHE,BLURING OF VISION & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL PARIETAL SOL | RIGHT FRONTO-PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 5X3X1 CM |
| 133 | 560730 | 10447-18 | MK | 55 | F | HEADACHE & RIGHT SIDED WEAKNESS | NA | EXCISED LEFT TEMPORAL MASS | LEFT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 8X4X2 CM |
| 134 | 560027 | 10508-18 | VS | 32 | M | HEADACHE,BLURING OF VISION & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL MASS | RIGHT FRONTAL MASS & BRAIN TISSUE | LOW GRADE GLIOMA | MULTIPLE 11X7X4.5 CM |
| 135 | 569257 | 10845-18 | PK | 39 | F | HEADACHE,DIZZINESS & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 5X3X1 CM |
| 136 | 561652 | 11497-18 | HS | 42 | M | HEADACHE,BLURING OF VISION & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL TUMOR | LEFT FRONTAL TUMOR | HIGH GRADE GLIOMA | MULTIPLE 5X3X1 CM |
| 137 | 578263 | 11522-18 | RT | 32 | F | HEADACHE & VOMITING | NA | LEFT FRONTAL - INTRA PARENCHYMAL CLOT | LEFT FRONTAL | NA | MULTIPLE 3X1.8X1.1 CM |
| 138 | 578888 | 11697-18 | MK | 53 | F | HEADACHE,BLURING OF VISION & MUSCLE WEAKNESS | K/C/O CA BREAST | FRONTAL CYSTIC LESION | RIGHT FRONTAL | METASTATIC CARCINOMA | SINGLE 3X2X1 CM |
| 139 | 583700 | 11765-18 | JK | 65 | F | HEADACHE,BLURING OF VISION & WEAKNESS OF ALL LIMBS | NA | LEFT FRONTAL MENINGIOMA | LEFT FRONTAL | MENINGIOMA | MULTIPLE 10X5X3 CM |
| 140 | 606610 | 13215-18 | IK | 52 | F | HEADACHE & BLURING OF VISION | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | LYMPHOMA | MULTIPLE 4.5X4X0.5 CM |
| 141 | 610277 | 13489-18 | BS | 55 | M | HEADACHE & RIGHT SIDED WEAKNESS | NA | LEFT PARIETAL SOL | LEFT PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 4X2X0.8 CM |
| 142 | 559101 | 13613-18 | BS | 55 | M | HEADACHE,BLURING OF VISION & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL TUMOR | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 6X4X1.5 CM |
| 143 | 617856 | 13942-18 | KK | 35 | F | HEADACHE, WEAKNESS OF MUSCLES & DIFFICULTY IN BALANCE WALKING | NA | POSTERIOR FOSSA MASS | POSTERIOR FOSSA | MENINGIOMA | MULTIPLE 4X2X1 CM |
| 144 | 621351 | 14072-18 | PK | 53 | M | HEADACHE,BLURING OF VISION & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | HYPERINTENCE LESION WITH EDEMA | MULTIPLE 1.8X1.5X0.6 CM |
| 145 | 616958 | 14163-18 | MS | 31 | M | HEADACHE & WEAKNESS OF MUSCLES | NA | FRONTAL SOL | LEFT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 8X5X3 CM |
| 146 | 613556 | 14198-18 | RS | 63 | M | HEADACHE & DIFFICULTY IN WRITING | NA | PARIETAL SOL | LEFT PARIETAL | MENINGIOMA | MULTIPLE 7.5X5X1.5 CM |
| 147 | 606863 | 14200-18 | SS | 56 | M | HEADACHE,DIZZINESS & RIGHT SIDED WEAKNESS | NA | LEFT FRONTO-PARIETAL SOL | LEFT FRONTO-PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 148 | 622838 | 14255-18 | RD | 56 | F | HEADACHE & BLURING OF VISION | NA | LEFT FRONTAL SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 149 | 622121 | 14365-18 | SS | 63 | M | HEADACHE,DIZZINESS & BLURING OF VISION | NA | FRONTAL SOL | LEFT FRONTAL | FRONTAL SOL | SINGLE 2.5X2X2 CM |
| 150 | 123340 | 14470-18 | BS | 74 | M | HEADACHE & DIZZINESS | NA | CEREBELLOPONTINE ANGLE SOL | CEREBELLOPONTINE ANGLE | BENIGN NEUROGENIC TUMOR | MULTIPLE 4X3X1 CM |
| 151 | 626019 | 14504-18 | RS | 57 | M | HEADACHE,BLURING OF VISION & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | SINGLE 5X4X2 CM |
| 152 | 401647 | 14853-18 | AS | 55 | M | SEVERE HEADACHE & DIZZINESS | K/C/O CA LUNG | BRAIN SOL | RIGHT FRONTAL | METASTATIC CARCINOMA | MULTIPLE 11X4.5X1.5 CM |
| 153 | 409958 | 14914-18 | N | 42 | M | SEVERE HEADACHE & WEAKNESS OF LIMBS | NA | BRAIN SOL | PARASAGITTAL | PARASAGITTAL SOL | SINGLE 3X2.5X1 CM |
| 154 | 638453 | 15782-18 | JK | 38 | M | HEADACHE,DIZZINESS & WEAKNESS | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 4X2.5X1 CM |
| 155 | 419097 | 86-19 | NK | 60 | F | INCOORDINATION ATAXIA | NA | CEREBELLAR SOL | CEREBELLAR | CEREBELLAR SOL | MULTIPLE 4X3X1 CM |
| 156 | 657906 | 736-19 | PP | 55 | M | HEADACHE | NA | INTRACRANIAL HEMATOMA | RIGHT FRONTAL | HYPERDENSE LESION WITH MASS EFFECT | MULTIPLE 2X1X0.5 CM |
| 157 | 661090 | 991-19 | RM | 71 | M | HEADACHE | H/O HEAD TRAUMA | SUBDURAL HEMATOMA | LEFT DURAL MEMBRANES | HYPERDENSE LESION WITH MASS EFFECT | SINGLE 5X3X0.5 CM |
| 158 | 662346 | 1078-19 | RS | 58 | M | HEADACHE & LEFT SIDED WEAKNESS | NA | GLIOBLASTOMA | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 3X2X1 CM |
| 159 | 660046 | 1136-19 | KK | 60 | F | VISUAL DEFECTS & HEADACHE | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 3X2X1 CM |
| 160 | 663281 | 1152-19 | DD | 58 | F | HEADACHE | NA | MENINGIOMA | LEFT FRONTO PARIETAL | LEFT FRONTO PARIETAL | MULTIPLE 6.5X3.5X1.2 CM |
| 161 | 654557 | 1356-19 | RK | 59 | M | HEADACHE & SEIZURES | NA | HIGH GRADE GLIOMA | RIGHT TEMPORAL | HIGH GRADE GLIOMA -? GLIOBLASTOMA | MULTIPLE 7X5X3 CM |
| 162 | 656486 | 1401-19 | AS | 54 | M | HEADACHE | NA | MENINGIOMA | LEFT TEMPORAL | LEFT TEMPORAL | MULTIPLE 10X6X2 CM |
| 163 | 563253 | 1453-19 | RK | 36 | M | SEIZURES | NA | SUB DURAL HEMORRHAGE | LEFT MEMBRANES | ?SUBDURAL HEMORRHAGE | MULTIPLE 5X4X2 CM |
| 164 | 673277 | 1829-19 | RK | 59 | F | HEADACHE & SEIZURES | NA | TEMPORAL SOL ? HIGH GRADE GLIOMA | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 6X3X2 CM |
| 165 | 680247 | 2228-19 | GS | 36 | M | HEADACHE & SEIZURES | NA | BRAIN SOL | LEFT FRONTAL | HYPERINTENCE LESION WITH EDEMA | MULTIPLE 5X3X2 CM |
| 166 | 681481 | 2394-19 | CS | 3 | M | FOCAL NEUROLOGICAL DEFICIT & SEIZURES | NA | SOL FOURTH VENTRICLE | FOURTH VENTRICLE | FOURTH VENTRICLE SOL | MULTIPLE 6X4X3 CM |
| 167 | 687529 | 2747-19 | DS | 36 | M | HEADACHE & LEFT SIDED WEAKNESS | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 3X2X1.5 CM |
| 168 | 449614 | 2749-19 | KK | 58 | F | HEADACHE,SEIZURES & LOWER LIMB WEAKNESS | K/C/O CA OVARY | BRAIN SOL | LEFT PARIETAL | LEFT PARIETAL SOL ?METASTATIC | MULTIPLE 3X2X0.8 CM |
| 169 | 694814 | 3279-19 | RS | 66 | M | HEADACHE & SEIZURES | NA | INTRACRANIAL HEMORRHAGE | RIGHT FRONTAL | HYPERINTENCE LESION WITH EDEMA | MULTIPLE 6X4X2 CM |
| 170 | 695646 | 3303-19 | CD | 48 | F | HEADACHE | NA | MENINGIOMA | LEFT FRONTAL | MENINGIOMA | MULTIPLE 5X3X2 CM |
| 171 | 696525 | 3304-19 | JK | 55 | F | HEADACHE & SEIZURES | NA | CYSTIC LESION | RIGHT FRONTOPARIETAL | RIGHT FRONTOPARIETAL CYST WITH MASS EFFECT | MULTIPLE 0.8X0.6X0.4 CM |
| 172 | 697253 | 3393-19 | BR | 66 | M | HEADACHE | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 1X0.8X0.6 CM |
| 173 | 711441 | 4425-19 | GS | 60 | M | HEADACHE & RIGHT SIDED WEAKNESS | NA | ? GLIOBLASTOMA | LEFT OCCIPITAL | HIGH GRADE GLIOMA -? GLIOBLASTOMA | MULTIPLE 4X3X2 CM |
| 174 | 713957 | 4666-19 | NS | 50 | M | HEADACHE | NA | CEREBELLAR SOL | CEREBELLAR | CEREBELLAR SOL | MULTIPLE 2X1X0.5 CM |
| 175 | 715942 | 4751-19 | R | 5 | F | HEADACHE & SEIZURES | NA | ? LOW GRADE GLIOMA | POSTERIOR FOSSA | ENHANCING CYSTIC AND SOLID LESION | MULTIPLE 6X4X2 CM |
| 176 | 720999 | 5087-19 | JS | 42 | M | HEADACHE | NA | INTRACRANIAL HEMORRHAGE | RIGHT TEMPORAL | NA | MULTIPLE 2X1X0.5 CM |
| 177 | 713058 | 5379-19 | SR | 60 | M | HEADACHE & SEIZURES | NA | GLIOBLASTOMA | RIGHT FRONTAL | HIGH GRADE GLIOMA -? GLIOBLASTOMA | MULTIPLE 6X4X1 CM |
| 178 | 748580 | 7318-19 | SS | 60 | M | LOSS OF CONCIOUSNESS | K/C/O HTN | VENOUS INFARCT | RIGHT TEMPOROPARIETAL | VENOUS INFARCT - BRAIN | MULTIPLE 2X1.5X1 CM |
| 179 | 749121 | 7354-19 | NR | 47 | F | GEN. WEAKNESS & SEIZURES | NA | INFARCT BRAIN | LEFT TEMPORAL | HYPERINTENCE LESION WITH EDEMA | SINGLE 1.2X0.8X0.2 CM |
| 180 | 753506 | 7914-19 | SB | 55 | F | HEADACHE & VERTIGO | NA | CEREBELLOPONTINE ANGLE MASS | CEREBELLOPONTINE ANGLE | BENIGN NEUROGENIC TUMOR | MULTIPLE 4X3X2 CM |
| 181 | 756882 | 8072-19 | RKG | 52 | M | HEADACHE & ATAXIA | NA | PERISYLVIAN SOL | RIGHT PERISYLVIAN | LOW GRADE GLIOMA | MULTIPLE 9X6X3 CM |
| 182 | 755915 | 8104-19 | KD | 56 | M | HEADACHE,SEIZURES & RIGHT SIDED WEAKNESS | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 6X5X4 CM |
| 183 | 748178 | 8396-19 | D | 7 | M | FEVER & SEIZURES | H/O CSOM | ? ABSCESS | LEFT FRONTAL | HYPERINTENCE LESION WITH EDEMA | MULTIPLE 0.8X0.5X0.4 CM |
| 184 | 768059 | 8876-19 | CS | 57 | M | SEVERE HEADACHE & FOCAL NEUROLOGICAL DEFECT | NA | MENINGIOMA | RIGHT & LEFT FRONTAL | MENINGIOMA | MULTIPLE 7X3X2 CM |
| 185 | 4579 | 9108-19 | SD | 55 | M | HEADACHE & SEIZURES | NA | HIGH GRADE GLIOMA | RIGHT TEMPOROPARIETAL | HIGH GRADE GLIOMA | MULTIPLE 6X5X4 CM |

| SR. NO. | MRD | MICROSCOPIC EXAMINATION | SPECIAL STAINS | | | | |
|---|---|---|---|---|---|---|---|
| | | | AFB | PAS | GMS | RETICULIN | PERL'S |
| 94 | 402201 | SECTIONS FROM BRAIN PARENCHYMA SHOW EXTENSIVE AREAS OF INTRACRANIAL HAEMORRHAGE WITH BLOOD CLOT. CYSTIC CHANGE OF BRAIN PARENCHYMA NOTED.NO EVIDENCE OF MALIGNANCY SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 95 | 401453 | UMOR COMPOSED OF SQUAMOUS EPITHELIAL CELLS ARRANGED IN BROAD STRANDS AND CORDS WHICH ARE BORDERED BY PALISADING COLUMNAR EPITHELIUM. NODULES OF ANUCLEATE SQUAMES (WET KERATIN) ALSO IDENTIFIED. MANY STELLATE CELLS WITHIN THE MICROCYTES IDENTIFIED. SURROUNDING BRAIN PARENCHYMA SHOWS NUMEROUS ROSENTHAL | NA | NA | NA | NA | NA |
| 96 | 402531 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 97 | 403644 | SECTION MAINLY SHOW FIBROCOLLAGENOUS TISSUE WITH MILD INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE ALONG WITH CHOLESTROL CLEFTS. AREAS OF HAEMORRHAGE AND NECROSIS ALSO SEEN. | NA | NA | NA | NA | NA |
| 98 | 401798 | GED IN DIFFUSE SHEETS. TUMOR CELLS HAVE MILD PLEOMORPHIC, VESICULAR NUCLEUS WITH SCANTY CYTOPLASM PRESENT AGAINST A FIBRILLARY BACKGROUND. SHEETS OF GEMISTIOCYTES HAVING ECCENTRIC PLACED NUCLEI AND MODERATE AMOUNT OF EOSINOPHILIC CYTOPLASM. FOCAL CYSTIC CHANGES IDENTIFIED. FEW TUMOR GIANT CELLS AND MITOTI | NA | NA | NA | NA | NA |
| 99 | 406646 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 100 | 389792 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 101 | 412409 | TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBIT MODERATE NUCLEAR PLEOMORPHISM WITH VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. IN FOCAL AREAS TUMOR CELLS ARE PALISADED AROUND AREAS OF NECROSIS. MITOTIC FIGURES NOTED. FOCAL AREA | NA | NA | NA | NA | NA |
| 102 | 418434 | H TUMOR CELLS ARRANGED IN LOBULES AND NESTS. INDIVIDUAL TUMOR CELLS ARE POLYGONAL WITH VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. FOCI OF NECROSIS IDENTIFIED. THERE IS DENSE LYMPHOCYTIC INFILTRATE AROUND THE TUMOUR NESTS. NORMAL BRAIN PARENCHYMA SHOWS INFILTRATION BY TUMOR CE | NA | NA | NA | NA | NA |
| 103 | 417775 | SECTIONS SHOW A WELL CIRCUMSCRIBED TUMOR WITH TUMOR CELLS PRESENT IN SYNCITIAL PATTERN. INDIVIDUAL TUMOR CELLS HAVE OVAL NUCLEI WITH DISPERSED CHROMATIN AND VARIABLE AMOUNT OF EOSINOPHILIC CYTOPLASM.NO SIGNIFICANT MITOSIS/NECROSIS SEEN IN SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 104 | 422241 | SECTIONS TAKEN FROM THE BRAIN SHOWS AREAS OF HAEMORRHAGE AND MANY DILATED AND CONGESTED BLOOD VESSELS. FEW OF THE VASCULAR CHANNELS HAVE IRREGULARLY THICKENED WALLS. | NA | NA | NA | NA | NA |
| 105 | 425016 | NCE OF CELLULAR TUMOR WITH CELLS ARRANGED IN DIFFUSE SHEETS PRESENT AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE UNIFORM ROUND NUCLEI WITH A CLEAR PERINUCLEAR HALO AROUND IT. MANY DELICATE BRANCHING CAPILLARIES NOTED. FEW GEMISTIOCYTES ALSO IDENTIFIED. NO SIGNIFICANT NUCLEAR ATYPIA/MITOSIS/NE | NA | NA | NA | NA | NA |
| 106 | 431144 | CKGROUND. INDIVIDUAL TUMOR CELLS EXHIBITING MINIMAL NUCLEAR PLEOMORPHISM WITH HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. PROMINENT MICROCYSTIC CHANGE NOTED. ALSO IDENTIFIED IS PERIVASCULAR CUFFING OF THE LYMPHOCYTES. THE TUMOR CELLS ARE INFILTRATING INTO ADJACENT BRAIN PARENCHYMA.AREAS OF | NA | NA | NA | NA | NA |
| 107 | 431378 | SECTIONS FROM THE BRAIN SHOW PRESENCE OF A CELLULAR TUMOR ON THE SURFACE OF BRAIN ARRANGED IN FORM OF SHEETS. INDIVIDUAL TUMOR CELLS EXHIBIT MILD TO MODERATE NUCLEAR PLEOMORPHISM, HYPERCHROMATIC NUCLEI AND SCANT CYTOPLASM. BRISK MITOSIS AND FOCI OF NECROSIS NOTED. THE TUMOR IS FOCALLY INFILTRATING THE ADJACENT BRAIN. | NA | NA | NA | NA | NA |
| 108 | 437630 | SECTIONS SHOW PRESENCE OF CELLULAR TUMOR WITH CELLS ARRANGED IN SYNCYTIAL PATTERN. INDIVIDUAL TUMOR CELLS HAVE OVAL NUCLEI, DELICATE CHROMATIN, INCONSPICUOUS NUCLEOLI AND ABUNDANT AMOUNT OF EOSINOPHILIC GRANULAR CYTOPLASM. TUMOR CELLS ARE INFILTRATING INTO THE BONE. | NA | NA | NA | NA | NA |
| 109 | 435876 | SECTION SHOW A CELLULAR TUMOR ARRANGED IN SHEETS AND NESTS SEPARATED BY THIN FIBROVASCULAR SEPTAE. INDIVIDUAL CELLS EXHIBIT MILD NUCLEAR PLEOMORPHISM AND BONE FINE NUCLEAR CHROMATIN AND MODERATE CYTOPLASM.NO SIGNIFICANT MITOSIS/NECROSIS SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | RETICULIN LOSS | NA |
| 110 | 457525 | HISTOLOGY CONSISTENT WITH BLOOD CLOT- BRAIN TISSUE.NO GRANULOMA/TISSUE SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 111 | 460964 | SECTIONS SHOW PRESENCE OF CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS, NODULES AND PERIVASCULAR ARRANGEMENT WHICH IS INFILTRATING THE PARENCHYMA. INDIVIDUAL TUMOR CELLS ARE ROUND HAVING VESICULAR NUCLEI, FINE CHROMATIN AND SCANT AMOUNT OF CYTOPLASM. APOPTOTIC BODIES, MITOSIS AND FOCI OF NECROSIS SEEN. | NA | NA | NA | NA | NA |
| 112 | 472333 | FROM BRAIN SHOW A WELL CIRCUMSCRIBED TUMOR ARRANGED IN GLANDS AND PAPILLAE. INDIVIDUAL TUMOR CELLS EXHIBIT MODERATE PLEOMORPHISM, HYPERCHROMATIC NUCLEI, INCONSPICOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. MANY BIZARRE AND MULTINUCLEATED GIANT CELLS SEEN. LARGE AREAS OF HAEMORRHAGE AND NECROSIS I | NA | NA | NA | NA | NA |
| 113 | 478141 | LAR AND HYPERCELLULAR AREAS. IN HYPERCELLULAR AREAS CELLS ARE ELONGATED HAVING SPINDLE SHAPED NUCLEI PRESENT AGAINST A FIBRILLARY BACKGROUND. OCCASIONAL ROSENTHAL FIBRES IDENTIFIED. IN THE HYPOCELLULAR AREAS ARE SEEN EOSINOPHILIC GRANULAR BODIES. MANY CONGESTED BLOOD VESSELS WITH HYALINIZATION OF WALL SEEN | NA | NA | NA | NA | NA |
| 114 | 485287 | SECTIONS SHOW DIFFUSE INFILTRATION OF A CELLULAR TUMOR. INDIVIDUAL TUMOR CELLS ARE LARGE IN SIZE, HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI, MODERATE AMOUNT OF CYTOPLASM AND EXHIBIT MODERATE TO MARKED PLEOMORPHISM. BIZARRE FORMS AND BRISK MITOSIS NOTED. NUMEROUS LYMPHOCYTES ARE SEEN IN THE BACKGROUND. | NA | NA | NA | NA | NA |
| 115 | 489703 | MOR WITH TUMOR CELLS ARRANGED IN FASCICLES. INDIVIDUAL TUMOR CELLS ARE SPINDLE SHAPED HAVING VESICULAR NUCLEI, PROMINENT NUCLEOLI AND ILL-DEFINED CYTOPLASMIC BOUNDARIES. OCCASIONAL PSAMMOMA BODIES NOTED. NO NECROSIS/SMALL CELLS/SHEETING ARRANGEMENT/SIGNIFICANT MITOSIS SEEN IN THE SECTIONS EXAMINED. NO BRAIN P | NA | NA | NA | NA | NA |
| 116 | 492864 | ESENCE OF CELLULAR TUMOR WITH EXTENSIVE AREAS OF TUMOR NECROSIS. TUMOR CELLS ARE ARRANGED IN DIFFUSE SHEETS PRESENT AGAINST A FIBRILLARY BACKGROUND. THESE CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. MANY MITOTIC FIGURES AND TUMOR GIANT CELLS IDENTIFIED. ENDOTHELIAL CELL PROLIFERATI | NA | NA | NA | NA | NA |
| 117 | 496018 | HISTOMORPHOLOGICAL FEATURES ARE CONSISTENT WITH BLOOD CLOT.NO EVIDENCE OF MALIGNANCY SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 118 | 496293 | OF A WELL CIRCUMSCRIBED CELLULAR TUMOR WITH CELLS ARRANGED IN DIFFUSE SHEETS AND FOCAL PERIVASCULAR ARRANGEMENT. THE CELLS HAVE VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI AND MODERATE TO ABUNDANT AMOUNT OF CYTOPLASM. FOCAL FIBRILLARY BACKGROUND NOTED. HOWEVER, NO SIGNIFICANT MITOSIS/NECROSIS/ENDOTHELIAL | NA | NA | NA | NA | NA |
| 119 | 464221 | SIONAL CELL HAVING HYPERCHROMATIC NUCLEUS.SECTIONS SHOW TAKEN FROM THE DEEP SHOWS PRESENCE OF NORMAL BRAIN PARENCHYMA WITH FOCAL AREAS SHOWING PRESENCE OF CELLS HAVING PLEOMORPHIC HYPERCHROMATIC NUCLEI PRESENT AGAINST A FIBRILLARY BACKGROUND. FEW TUMOR GIANT CELLS IDENTIFIED. AN OCCASIONAL MITOTIC F | NA | NA | NA | NA | NA |
| 120 | 444920 | ARRANGED IN FASCICLES AND WHORLS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, INCONSPICUOS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM WITH INDISTINCT CELL BOUNDARIES. FEW PSAMMOMA BODIES ALSO SEEN. HOWEVER, NO NECROSIS/SMALL CELL CHANGE/SHEETING PATTERN/SIGNIFICANT MITOSIS IDENTIFIED IN THE SECTIONS EXAM | NA | NA | NA | NA | NA |
| 121 | 502444 | ONS TAKEN FROM BRAIN SHOW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEUS. MANY MITOTIC FIGURES NOTED. FOCI OF HAEMORRHAGE AND NECROSIS ALSO NOTED.THE TUMOR CELLS ARE INFILTRATING INTO THE ADJACENT PARENC | NA | NA | NA | NA | NA |
| 122 | 505064 | SECTIONS SHOW FIBROCOLLAGENOUS TISSUE WITH INFILTRATION BY TUMOR CELLS PRESENT IN GLANDS WITH FOCAL PAPILLARY CONFIGURATION. THE INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI PROMINENT NUCLEOLI AND MODERATE CYTOPLASM WITH NUCLEAR STRATIFICATION NOTED AT PLACE. | NA | NA | NA | NA | NA |
| 123 | 516719 | SECTIONS SHOW INFILTRATION OF THE BRAIN PARENCHYMA BY A CELLULAR TUMOR WITH INDIVIDUAL TUMOR CELLS HAVING HYPERCHROMATIC NUCLEI, INCONSPICOUS NUCLEOLI AND SCANT AMOUNT OF CYTOPLASM.TINY FOCI OF NECROSIS AND ENDOTHELIAL CELL PROLIFERATION NOTED. ATYPICAL BRISK MITOSIS SEEN. | NA | NA | NA | NA | NA |
| 124 | 532310 | SECTION SHOW AREAS OF HAEMORRHAGE WITH NEUTROPHILIC INFILTRATE.HOWEVER, NO GRANULOMA/EVIDENCE OF MALIGNANCY SEEN IN THE SECTION EXAMINED. | NA | NA | NA | NA | NA |
| 125 | 531353 | GED IN WHORLS AND SYNCITIUM. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, CONSPICUOUS COARSE NUCLEI AND MODERATE AMOUNT OF CYTOPLSM WITH INDISTINCT CELL BOUNDARIES.HOWEVER, NO SIGNIFICANT MITOSIS/NECROSIS/SHEETING PATTERN/SMALL CELL CHANGE NOTED IN THE SECTIONS EXAMINED.TUMOR IS INFILTRATING THE BONE AND IS | NA | NA | NA | NA | NA |
| 126 | 532909 | OF TUMOR WITH TUMOR CELLS ARRANGED IN SYNCYTIAL PATTERN AND WHORLS. INDIVIDUAL TUMOR CELLS HAVE ROUND TO OVAL VESICULAR NUCLEI, INCONSPICUOUS NUCLEOLI AND INDISTINCT CYTOPLASMIC BOUNDARIES.TINY BIT OF NORMAL BRAIN PARENCHYMA DOES NOT SHOW INVASION BY TUMOR.NO SHEETING PATTEN/MITOSIS/NECROSIS SEEN IN THE MI | NA | NA | NA | NA | NA |
| 127 | 531502 | E AREAS OF NECROSIS WITH PRESENCE OF CELLULAR TUMOR ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS EXHIBIT NUCLEAR PLEOMORPHISM WITH HYPERCHROMASIA AND PRESENCE OF MITOTIC FIGURES. BLOOD VESSELS SHOW PERIVASCULAR LYMPHOCYTIC INFILTRATE. AREAS OF HEMORRHAGE WITH HEMOSIDERIN LADEN MACROPHAGES NOTED. ENDO | NA | NA | NA | NA | NA |
| 128 | 541231 | OR RRANGED IN WHORLS AND SYNCYTIUM.INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, INCONSPICOUS NUCLEOLI AND SCANT CYTOPLASM. FEW OF THE CELLS SHOW INTRANUCLEAR INCLUSIONS AS WELL.MANY PSAMMOMA BODIES ALSO IDENTIFIED.HOWEVER, NO SHEETING PATTERN/NECROSIS/ATYPICAL MITOSIS SEEN IN SECTIONS EXAMINED.NO BRAIN P | NA | NA | NA | NA | NA |
| 129 | 542975 | SECTIONS MAINLY SHOW BLOOD CLOT, FOCAL ACUTE AND CHRONIC INFLAMMATORY INFILTRATE. NO EVIDENCE OF MALIGNANCY SEEN IN MULTIPLE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 130 | 542798 | NGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND . THERE IS PRESENCE OF NECROSIS AROUND WHICH THERE IS PALISADING OF TUMOR CELLS. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEI AND ILL DEFINED CYTOPLASMIC BOUNDARIES. THERE IS ENDOTHELIAL CELL PROLIFERATION ALONG WITH PRESENCE OF MITOTIC FIGURES. | NA | NA | NA | NA | NA |
| 131 | 545674 | E SHEETS PRESENT AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. FEW OF THE CELLS ARE ARRANGED IN FASCICLES WITH CELLS HAVING SPINDLE SHAPED NUCLEI AND ILL DEFINED CYTOPLASM.BIZARRE TUMOR CELLS IDENTIFIED. ENDOTHELIAL CELL PROLIFERATION SE | NA | NA | NA | NA | NA |
| 132 | 506598 | RRANGED IN NESTS AGAINST A FOCAL FIBRILLARY BACKGROUND. THE TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEI WITH ILL-DEFINED CYTOPLASMIC BOUNDARIES.THE TUMOR SHOWS PERIVASCULAR ARRANGEMENT AS WELL. FOCAL ENDOTHELIAL CELL PROLIFERATION IDENTIFIED,ATYPICAL MITOTIC FIGURES AND LARGE AREA OF HAEMORRHAGE | NA | NA | NA | NA | NA |
| 133 | 560730 | S AND CONCENTRATED AROUND THE BLOOD VESSELS AGAINST FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI AND SCANT AMOUNT OF CYTOPLASM. MANY GEMISTIOCYTES NOTED.EXTENSIVE ENDOTHELIAL CELL PROLIFERATION FORMING GLOMERULOID STRUCTURE SEEN. LARGE AREAS OF NECROSIS WITH TUMOR CELLS SE | NA | NA | NA | NA | NA |
| 134 | 560027 | W PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS ARE ROUND WITH HYPERCHROMATIC NUCLEI AND PERINUCLEAR HALO. ALSO SEEN ARE MANY CHICKEN WIRE VESSELS.PERINEURAL SATELLITOSIS AS WELL AS OCCASIONAL DYSTROPHIC CALCIFICATION NOTED.HOWEVER, NO NECROSIS/SIGNIFICANT MITOSIS/END | NA | NA | NA | NA | NA |
| 135 | 569257 | E SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. PERINUCLEAR CLEARING SEEN IN MANY OF THE CELLS.THE TUMOR CELLS ARE SEEN INFILTRATING INTO THE ADJACENT BRAIN PARENCHYMA.MANY REACTIVE ASTROCYTES ALSO NOTED. PROMINENT CYSTIC CHANGE ALSO IDENTIFIED.NO NECR | NA | NA | NA | NA | NA |
| 136 | 561652 | ND PERINUCLEAR HALO. MANY ARBORZING THIN CAPILLARIES – CHICKEN WIRE PATTERN OF VASCULATURE NOTED IN BACKGROUND.LARGE AREA OF CALCIFICATION NOTED. FOCI OF CYSTIC CHANGE NOTED. THE TUMOR IS INFILTRATING ADJACENT BRAIN PARENCHYMA. IN TWO SECTIONS EXAMINED TUMOR CELLS ARE SEEN IN DIFFUSE SHEETS HAVING A FIBRILLARY | NA | NA | NA | NA | NA |
| 137 | 578263 | SECTION SHOW EXTENSIVE AREA OF HEMORRHAGE WITH PRESENCE OF BLOOD CLOT. NO EVIDENCE OF MALIGNANCY SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 138 | 578888 | TIONS SHOW A WELL CIRCUMSCRIBED CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. LARGE AREAS OF NECROSIS SEEN. IN FOCAL AREA TUMOR CELLS ARE INFILTRATING THE BRAIN PARENCHYMA.NORMAL BRAIN PARENCHYMA SE | NA | NA | NA | NA | NA |
| 139 | 583700 | H TUMOR CELLS ARRANGED IN FASCICLES AND WHORLS. INDIVIDUAL TUMOR CELLS HAVE OVAL TO SPINDLE SHAPED NUCLEI AND SCANTY TO MODERATE AMOUNT OF EOSINOPHILIC CYTOPLASM HAVING INDISTINCT BOUNDARIES. OCCASIONAL PSAMMOMA BODIES SEEN. NO SHEETING PATTERN/NUCLEAR ATYPIA/MITOSIS SEEN IN THE MULTIPLE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 140 | 606610 | SECTIONS SHOW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN DIFFUSE SHEETS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND SCANT TO MODERATE AMOUNT OF CYTOPLASM. STARRY SKY APPEARANCE NOTED.BRISK MITOSIS SEEN. | NA | NA | NA | NA | NA |
| 141 | 610277 | OW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND ILL-DEFINED CYTOPLASM. TINY AREAS OF NECROSIS SEEN. LARGE AREA OF HEMORRHAGE ALSO IDENTIFIED. MANY MITOTIC FIGURES NOTED.TUMOR IS INFILTRAT | NA | NA | NA | NA | NA |
| 142 | 559101 | R WITH TUMOR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBIT MARKED PLEOMORPHISM AND HAVE VESICULAR PLEOMORPHIC NUCLEI.TUMOR GIANT CELLS NOTED. AREAS OF NECROSIS AND ENDOCAPILLARY CELL PROLIFERATION ALSO SEEN. CHICKENWIRE VASCULATION ALSO NOTED. BRISK MITOSIS SEEN. | NA | NA | NA | NA | NA |
| 143 | 617856 | ULAR WITH CELLS PRESENT IN FASCICLES AND SYNCYTIAL PATTERN. INDIVIDUAL TUMOR CELLS HAVE OVAL NUCLEI WITH DELICATE CHROMATIN AND MODERATE AMOUNT OF ILL-DEFINED CYTOPLASM. FEW PSAMMOMA BODIES SEEN. HOWEVER, NO SMALL CELL CHANGE/SHEETING/NECROSIS/ATYPICAL MITOSIS SEEN IN THE SECTIONS EXAMINED. NO BRAIN PARENCHY | NA | NA | NA | NA | NA |
| 144 | 621351 | SECTION TAKEN FROM THE BRAIN SHOW THICKENED AND DILATED BLOOD VESSELS ALONG WITH LARGE AREAS OF HAEMORRHAGE. FOCAL CYSTIC CHANGE ALSO IDENTIFIED.NO TUMOR IDENTIFIED IN THE MULTIPLE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 145 | 616958 | R CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND NUCLEI WITH PERINUCLEAR HALO. FOCALLY THIN WALL THICKEN WIRE VASCULATURE SEEN IN THE BACKGROUND. PROMINENT CYSTIC CHANGE ALSO NOTED. THE TUMOR CELLS INFILTRATING THE ADJACENT BRAIN PARENCHYMA WITH PERINEURONAL SATELLISM SEEN. HOWEVER, NO NUC | NA | NA | NA | NA | NA |
| 146 | 613556 | LS PRESENT IN WHORLS AND ISLANDS. INDIVIDUAL TUMOR CELLS HAVE ROUND TO OVAL NUCLEI WITH DELICATE CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. CYSTIC CHANGE AND THICK WALLED HYALINISED BLOOD VESSELS WALLS IDENTIFIED.ADJACENT BRAIN PARENCHYMA SHOWS NO TUMOR INFILTRATION. HOWEVER, NO ATYPIA/MITOSIS/NECROSIS/SI | NA | NA | NA | NA | NA |
| 147 | 606863 | WITH CELLS ARRANGED IN SHEETS AND ALSO PRESENT AROUND THE BLOOD VESSELS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. EXTENSIVE ENDOTHELIAL CELL PROLIFERATION NOTED. LARGE AREA OF GEOGRAFICAL NECROSIS WHICH IS PALISADED BY TUMOR CE | NA | NA | NA | NA | NA |
| 148 | 622838 | ULAR TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEI AND ILL DEFINED CYTOPLASMIC BOUNDARIES. THERE IS ALSO PRESENCE OF NECROSIS AND ENDOTHELIAL CELL PROLIFERATION. ATYPICAL MITOTIC FIGURES ALSO NOTED. TUMOR CELL INFILTRATING | NA | NA | NA | NA | NA |
| 149 | 622121 | SECTIONS TAKEN FROM BRAIN SHOW EXTENSIVE AREA OF NECROSIS SURROUNDED BY EPITHELIOID CELL GRANULOMAS, LYMPHOCYTES AND LANGHANS GIANT CELLS. | NEGATIVE | NA | NA | NA | NA |
| 150 | 123340 | SECTIONS SHOW PRESENCE OF A CELLULAR TUMOR SHOWING HYPERCELLULAR AND HYPOCELLULAR AREAS. THE TUMOR CELLS EXHIBIT MILD NUCLEAR PLEOMORPHISM HAVE SPINDLE SHAPED CELL WITH WAVY NUCLEI, INCONSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF ILL-DEFINED CYTOPLASM. CYSTIC CHANGE AND THICK WALL HYALINISED BLOOD VESSELS ALSO SEEN. NO SIGNIFICANT MIVI ELAR PLEOMORPHISM/MITOSIS/NECROSIS SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 151 | 626019 | AINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBIT MARKED PLEOMORPHISM AND HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM.MANY MULTINUCLEATED CELLS AND GEMISTOCYTES SEEN. LARGE AREAS OF NECROSIS PALISADED BY TUMOR CELLS AT PLACES NOTED. ENDOTHELIAL CELL PROLIFERATION EVIDENT. F | NA | NA | NA | NA | NA |
| 152 | 401647 | SECTION SHOW PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN PAPILLAE GLANDS AND NESTS. THESE PAPILLAE ARE LINED BY PLEOMORPHIC CELLS HAVING HYPERCHROMATIC NUCLEUS AND MODERATE AMOUNT OF CYTOPLASM. LARGE AREAS OF NECROSIS AND BRISK MITOSIS NOTED. | NA | NA | NA | NA | NA |
| 153 | 409958 | F A WELL CIRCUMSCRIBED CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE OVAL TO SPINDLE SHAPED NUCLEI, CONSPICIOUS NUCLEOLI AND SCANT AMOUNT OF SLIGHTLY EOSINOPHILIC CYTOPLASM. THESE CELLS ARE FORMING AGGREGATES AROUND BLOOD VESSELS.MITOSIS <5/10 HPF NOTED. HOWEVER, NO NECROSIS SE | NA | NA | NA | NA | NA |
| 154 | 638453 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | RETICULIN LOSS | NA |
| 155 | 419097 | EN FROM BRAIN SHOW PRESENCE OF WELL CIRCUMSCRIBED TUMOR WITH TUMOR CELLS ARRANGED IN GLANDS AND PAPILLAE. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEUS, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM ALONG WITH AREA OF NECROSIS AND HEMORRHAGE. THE TUMOR CELLS ARE INFILTRATING THE ADJACENT BRAIN | NA | NA | NA | NA | NA |
| 156 | 657906 | SECTIONS MAINLY SHOW BLOOD CLOT WITH THICKENED AND DILATED VASCULAR CHANNELS.TINY BRAIN PARENCHYMA SHOWING CONGESTION SEEN. NO TUMOR IDENTIFIED IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 157 | 661090 | SECTIONS SHOW MEMBRANOUS TISSUE HAVING FIBROCOLLAGENOUS TISSUE CONTAINING CONGESTED VESSELS AND MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS WITH FOAMY MACROPHAGES. BLOOD CLOT WITH PRESENCE OF MENIGOTHELIAL CELLS. | NA | NA | NA | NA | NA |
| 158 | 662346 | AINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBIT MARKED PLEOMORPHISM AND HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM.MANY MULTINUCLEATED CELLS AND GEMISTOCYTES SEEN. LARGE AREAS OF NECROSIS PALISADED BY TUMOR CELLS AT PLACES NOTED. ENDOTHELIAL CELL PROLIFERATION EVIDENT. F | NA | NA | NA | NA | NA |
| 159 | 660046 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | NA | NA |
| 160 | 663281 | OW A CELLULAR TUMOR WITH CELLS ARRANGED IN WHORLS. INDIVIDUAL TUMOR CELLS EXHIBIT MILD NUCLEAR PLEOMORPHISM, VESICULAR NUCLEI, INCONSPICOUS NUCLEOLI.FOCAL AREA SHOWS ADIPOCYTES. ALSO SEEN ARE PSAMMOMA BODIES IN THE SECTIONS EXAMINED.NO SHEETING PATTERN/SMALL CELL CHANGE/MITOSIS/NECROSIS SEEN IN THE SECTIO | NA | NA | NA | NA | NA |
| 161 | 654557 | OW PRESENCE OF CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBIT MODERATE PLEOMORPHISM, HYPERCHROMATIC NUCLEI AND HAVE MODERATE AMOUNT OF CYTOPLASM. AREAS OF NECROSIS SEEN. MITOTIC FIGURES NOTED. TUMOR IS INFILTRATING THE ADJACENT NORMAL BRAIN P | NA | NA | NA | NA | NA |
| 162 | 656486 | R TUMOR WITH TUMOR CELLS ARRANGED IN WHORLS, FASCICLES AND SYNCITIAL PATTERN. INDIVIDUAL TUMOR CELLS HAVE OVAL NUCLEI WITH VESICULAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CYTOPLASMIC BOUNDARIES. FEW CELLS HAVE NUCLEAR PSEUDOINCLUSIONS. HOWEVER, NO SHEETING/MITOSIS/NECROSIS SMALL CE | NA | NA | NA | NA | NA |
| 163 | 563253 | SECTIONS SHOW FIBROCOLLAGENOUS TISSUE WITH AREAS OF HEMORRHAGE ALONG WITH CHRONIC INFLAMMATORY INFILTRATE AND HEMOSIDERIN LADEN MACROPHAGES.NO EVIDENCE OF MALIGNANCY SEEN IN THE SECTIONS EXAMINED. | NA | NA | NA | NA | NA |
| 164 | 673277 | A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. LARGE AREAS OF PALISADING NECROSIS NOTED.MANY GEMISTOCYTES ALSO SEEN. ENDOTHELIAL CELL PROLIFERATION AND ATYPICAL MITOTIC FIGURES NOTED. MANY AREAS OF DYSTROPHIC CALCIFICATION SEEN. TUMOR | NA | NA | NA | NA | NA |
| 165 | 680247 | SECTIONS TAKEN FROM BRAIN SHOWS PRESENCE OF NUMEROUS DILATED AND CONGESTED BLOOD VESSELS, FEW OF WHICH HAVE VARIABLE THICKNESS OF THEIR WALL- SUGGESTIVE OF ARTERIO- VENOUS MALFORMATION. | NA | NA | NA | NA | NA |
| 166 | 681481 | PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN DIFFUSE SHEETS AND PALISADED AROUND VASCULAR CHANNELS FORMING PSEUDOROSETTES. TRUE ROSETTES ALSO IDENTIFIED. TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI WITH MODERATE CYTOPLASM AND FIBRILLARY BACKGROUND. AREAS OF NECROSIS AND HEMORRHAGE | NA | NA | NA | NA | NA |
| 167 | 687529 | CTIONS FROM THE BRAIN SHOW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN DIFFUSE SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND VESICULAR NUCLEI WITH PERINUCLEAR HALO. CHICKEN-WIRE VASCULATURE NOTED. PERINEURONAL SATELLITOSIS ALSO SEEN,THE TUMOR IS INFILTRATING THE ADJACENT NORMAL BRAIN PARENCHYM | NA | NA | NA | NA | NA |
| 168 | 449614 | SECTIONS TAKEN FROM BRAIN SHOWS PRESENCE OF A WELL CIRCUMSCRIBED CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN NESTS, SHEETS AND FOCAL GLANDULAR CONFIGURATION.INDIVIDUAL TUMOR CELLS SHOW PLEOMORPHIC VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. EXTENSIVE AREAS OF NECROSIS SEEN. MARKED NUCLEAR ATYPIA WITH ATYPICAL MITOTIC FIGURES SEEN.NO INDIVIDUAL CELL KERATINISATION/FIBRILLARY BACKGROUND IDENT

| SR. NO. | MRD | CONGO RED | IMMUNO-HISTOCHEMISTRY | | | | | | | | | FINAL HISTOPATHOLOGICAL DIAGNOSIS | GRADE | IDH STATUS | CATEGORY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | GFAP | ATRX | IDH-1 | MIB-1 | P53 | CK | EMA | S100 | OTHER | | | | |
| 94 | 402201 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | INTRAPARENCHYMAL HEMORRHAGE | NA | NA | NON-NEOPLASTIC |
| 95 | 401453 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | CRANIOPHARYNGIOMA | I | NA | NEOPLASTIC |
| 96 | 402531 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 97 | 403644 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PACHYMENINGITIS | NA | NA | NON-NEOPLASTIC |
| 98 | 401798 | NA | POSITIVE | NA | NA | 20% | NA | NA | NA | NA | LCA - NEGATIVE | ANAPLASTIC ASTROCYTOMA | III | NA | NEOPLASTIC |
| 99 | 406646 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 100 | 389792 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 101 | 412409 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 102 | 418434 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 103 | 417775 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | I | NA | NEOPLASTIC |
| 104 | 422241 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 105 | 425016 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 106 | 431144 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 107 | 431378 | NA | NEGATIVE | NA | NA | 70% | NA | NEGATIVE | NEGATIVE | NA | VIMENTIN, CD99 - POSITIVE & LCA, SYNAPTOPHYSIN, CHROMOGRANIN-A, MYOGENIN, DESMIN, TDT - NEGATIVE | PRIMITIVE NEUROECTODERMAL TUMOR (PNET) | NA | NA | NEOPLASTIC |
| 108 | 437630 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | I | NA | NEOPLASTIC |
| 109 | 435876 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 110 | 457525 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 111 | 460964 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | NON HODGKIN LYMPHOMA - DLBCL | NA | NA | NEOPLASTIC |
| 112 | 472333 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 113 | 478141 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PILOCYTIC ASTROCYTOMA | I | NA | NEOPLASTIC |
| 114 | 485287 | NA | NEGATIVE | NA | NA | 80% | NA | NA | POSITIVE | NA | PAX5, CD10, CD20, MUM1, CD3- POSITIVE & PAN-CK, CD30, BCL6, CD138, ALK1- NEGATIVE | NHL- B CELL- ANAPLASTIC LARGE CELL TYPE(ALK NEGATIVE) | NA | NA | NEOPLASTIC |
| 115 | 489703 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 116 | 492864 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 117 | 496018 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 118 | 496293 | NA | POSITIVE | NA | NA | 7% | NA | NA | NEGATIVE | POSITIVE | VIMENTIN - POSITIVE & SYNAPTOPHYSIN - NEGATIVE | ASTROBLASTOMA | NA | NA | NEOPLASTIC |
| 119 | 464221 | NA | POSITIVE | NA | NA | 1% | NEGATIVE | NA | NA | POSITIVE | VIMENTIN - POSITIVE & SYNAPTOPHYSIN - NEGATIVE | PLEOMORPHIC XANTHOASTROCYTOMA | II | NA | NEOPLASTIC |
| 120 | 444920 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 121 | 502444 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 122 | 505064 | NA | NA | NA | NA | NA | NA | NA | NA | NA | CK7, WT1, CEA - POSITIVE & ER, TTF1, CDX2, MAMMAGLOBIN, NAPSIN-A - NEGATIVE | METASTATIC CARCINOM,ATOUS DEPOSITS - LIKELY PANCREATIC AND UPPER GIT | NA | NA | NEOPLASTIC |
| 123 | 516719 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 124 | 532310 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | INTRAPARENCHYMAL HEMORRHAGE | NA | NA | NON-NEOPLASTIC |
| 125 | 531353 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 126 | 532909 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | I | NA | NEOPLASTIC |
| 127 | 531502 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 128 | 541231 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 129 | 542975 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 130 | 542798 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 131 | 545674 | NA | POSITIVE | NA | NA | NA | NA | NA | NEGATIVE | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 132 | 506598 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 133 | 560730 | NA | POSITIVE | NA | NA | 15% | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 134 | 560027 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 135 | 569257 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 136 | 561652 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC OLIGOASTROCYTOMA | III | NA | NEOPLASTIC |
| 137 | 578263 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 138 | 578888 | NA | NA | NA | NA | 70% | NA | NA | NA | NA | ER, PR, HER-2/NEU - NEGATIVE | METASTATIC CARCINOMATOUS DEPOSITS - PRIMARY BREAST | NA | NA | NEOPLASTIC |
| 139 | 583700 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 140 | 606610 | NA | NA | NA | NA | 80% | NA | NA | NA | NA | LCA, CD20, MUM1, BCL2, BCL6 - POSITIVE & CD3, CD10, PAN-CK - NEGATIVE | NON HODGKIN LYMPHOMA - DLBCL | NA | NA | NEOPLASTIC |
| 141 | 610277 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 142 | 559101 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 143 | 617856 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 144 | 621351 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 145 | 616958 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 146 | 613556 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 147 | 606863 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 148 | 622838 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 149 | 622121 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TUBERCULOSIS | NA | NA | NON-NEOPLASTIC |
| 150 | 123340 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 151 | 626019 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 152 | 401647 | NA | NA | NA | NA | NA | NA | NA | NA | NA | CK7,EMA,TTF-1,NAPSIN-A POSITIVE & CK-20 NEGATIVE | METASTATIC CARCINOMATOUS DEPOSITS LIKELY FROM LUNG | NA | NA | NEOPLASTIC |
| 153 | 409958 | NA | NA | NA | NA | NA | NA | NA | NA | NA | HEMANGIOPERICYTOMA | HAEMANGIOPERICYTOMA | III | NA | NEOPLASTIC |
| 154 | 638453 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 155 | 419097 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 156 | 657906 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 157 | 661090 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPIDURAL/SUBDURAL HEMATOMA | NA | NA | NON-NEOPLASTIC |
| 158 | 662346 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 159 | 660046 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 160 | 663281 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METAPLASTIC MENINGIOMA | I | NA | NEOPLASTIC |
| 161 | 654557 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 162 | 656486 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 163 | 563253 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SUBDURAL HEMORRHAGE | NA | NA | NON-NEOPLASTIC |
| 164 | 673277 | NA | POSITIVE | NA | NA | 30% | NA | NA | NEGATIVE | NA | SYNAPTOPHYSIN - NEGATIVE | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 165 | 680247 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 166 | 681481 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | EPENDYMOMA | II | NA | NEOPLASTIC |
| 167 | 687529 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 168 | 449614 | NA | NEGATIVE | NA | NA | 85% | NA | NA | NA | NA | PAN CK, CK7 PAX8, P16, WT1, B-CATENIN, ER - POSITIVE & LCA, P63, CK20,CDX2, MAMAGLOBIN, TTF1- NEGATIVE | METASTATIC CARCINOMATOUS DEPOSITS - LIKELY HIG GRADE SEROUS CA OVARY | NA | NA | NEOPLASTIC |
| 169 | 694814 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 170 | 695646 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 171 | 696525 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARACHNOID CYST | NA | NA | NON-NEOPLASTIC |
| 172 | 697253 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 173 | 711441 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 174 | 713957 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | HAEMANGIOBLASTOMA | I | NA | NEOPLASTIC |
| 175 | 715942 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PILOCYTIC ASTROCYTOMA | I | NA | NEOPLASTIC |
| 176 | 720999 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | INTRAPARENCHYMAL HEMORRHAGE | NA | NA | NON-NEOPLASTIC |
| 177 | 713058 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 178 | 748580 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 179 | 749121 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 180 | 753506 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | SCHWANNOMA | I | NA | NEOPLASTIC |
| 181 | 756882 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 182 | 755915 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC EPENDYMOMA | III | NA | NEOPLASTIC |
| 183 | 748178 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ABSCESS - NON SPECIFIC | NA | NA | NON-NEOPLASTIC |
| 184 | 768059 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 185 | 4579 | NA | POSITIVE | POSITIVE | NEGATIVE | 40% | NA | NEGATIVE | NEGATIVE | NEGATIVE | PAN CK, HMB-45 - NEGATIVE | GLIOSARCOMA | IV | WILD TYPE | NEOPLASTIC |

| SR. NO. | MRD | HISTO NO. | NAME | AGE | SEX | CHIEF COMPLAINT | PAST HISTORY | CLINICAL DIAGNOSIS | SITE OF BIOPSY | RADIOLOGY | GROSS EXAMINATION OF SPECIMEN |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 186 | 773387 | 9214-19 | NS | 50 | F | HEADACHE & VISUAL DEFECTS | NA | PITUITARY MACROADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 2X1.5X1 CM |
| 187 | 771609 | 9252-19 | BS | 60 | M | HEADACHE & RIGHT SIDED WEAKNESS | NA | HIGH GRADE GLIAL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 6X5X4.5 CM |
| 188 | 782242 | 9781-19 | JS | 48 | M | SEVERE HEADACHE & SEIZURES | K/C/O GANGLIOGLIOMA | GANGLIOGLIOMA | RIGHT TEMPOROPARIETAL | HYPODENCE CALCIFIED MASS | MULTIPLE 4X3X2 CM |
| 189 | 775656 | 9871-19 | APS | 20 | M | SEIZURES & NEUROLOGICAL DEFECT | NA | POSTERIOR FOSSA TX | POSTERIOR FOSSA | HYPERDENSE MASS IN POSTERIOR FOSSA | MULTIPLE 6X3X2 CM |
| 190 | 782229 | 9931-19 | SD | 60 | F | HEADACHE,SEIZURES & RIGHT SIDED WEAKNESS | NA | ? GLIOBLASTOMA | RIGHT FRONTAL | LEFT FRONTAL - GLIOBLASTOMA | MULTIPLE 5X4X3 CM |
| 191 | 785937 | 10011-19 | SP | 62 | M | HEADACHE | NA | MENINGIOMA | LEFT FRONTOPARIETAL | MENINGIOMA | MULTIPLE 7X5X2 CM |
| 192 | 788160 | 10107-19 | CK | 61 | F | SEIZURES & LEFT SIDED WEAKNESS | NA | ? GLIOBLASTOMA | RIGHT FRONTAL | HIGH GRADE GLIOMA - GLIOBLASTOMA | MULTIPLE 5X4X3 CM |
| 193 | 789534 | 10213-19 | AS | 61 | M | HEADACHE & SEIZURES | NA | ? HIGH GRADE GLIOMA | RIGHT FRONTAL | HIGH GRADE GLIOMA - GLIOBLASTOMA | MULTIPLE 4X2X2 CM |
| 194 | 792537 | 10467-19 | KS | 40 | M | HEADACHE, SEIZURES & ATAXIA | NA | LEFT CEREBELLAR SOL | LEFT CEREBELLUM | NON ENHANCING CYSTIC LESION | SINGLE 0.8X0.4X0.2 CM |
| 195 | 796026 | 10601-19 | SP | 63 | M | HEADACHE & FOCAL NEUROLOGICAL DEFECT | NA | RIGHT TEMPORAL SOL ? GLIOBLASTOMA | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 5X3.1X1.5 CM |
| 196 | 796346 | 10718-19 | GS | 64 | M | ACUTE ONSET PSYCHOSIS | NA | SOL OLFACTORY GROOVE | OLFACTORY GROOVE | OLFACTORY GROOVE MENINGIOMA | MULTIPLE 10X7X5 CM |
| 197 | 801209 | 10960-19 | UP | 54 | F | HEADACHE | NA | MENINGIOMA | RIGHT FRONTAL | MENINGIOMA | MULTIPLE 8X5X2 CM |
| 198 | 405782 | 11174-19 | VR | 51 | F | HEADACHE & SEIZURES | K/C/O CA BREAST | LEFT TEMPORAL SOL | LEFT TEMPORAL | METASTATIC CARCINOMA | MULTIPLE 6X5X4 CM |
| 199 | 811690 | 11714-19 | CS | 59 | M | HEADACHE & SEIZURES | K/C/O CA LUNG | ? METASTESIS | SUPERFICIAL & DEEP RIGHT TEMPORAL | RIGHT TEMPORAL SOL | MULTIPLE 9.5X7X4.5 CM |
| 200 | 813699 | 11872-19 | JS | 22 | M | HEADACHE & VISUAL DEFECTS | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 1.5X1X0.5 CM |
| 201 | 816047 | 12084-19 | RK | 47 | M | HEADACHE, FEVER & WEAKNESS | NA | MENINGIOMA | RIGHT MEMBRANES | MENINGIOMA | MULTIPLE 5X3X1 CM |
| 202 | 827318 | 12623-19 | R | 41 | F | HEADACHE & NEUROLOGICAL DEFICIT | NA | ? LOW GRADE GLIOMA | RIGHT FRONTOPARIETAL | LOW GRADE GLIOMA | MULTIPLE 8X6X5 CM |
| 203 | 815469 | 12683-19 | PK | 57 | F | HEADACHE & VISUAL DISTURBANCE | NA | LEFT OCCIPITAL SOL | LEFT OCCIPITAL | SOL IN LEFT OCCIPITAL | MULTIPLE 5.5X4X2 CM |
| 204 | 838215 | 13468-19 | V | 55 | F | HEADACHE & VISUAL DISTURBANCE | NA | PITUITARY MACROADENOMA | EXCISE PITUTARY MASS | PITUITARY MACROADENOMA | MULTIPLE 3X1.5X0.8 CM |
| 205 | 841193 | 13525-19 | BK | 38 | F | HEADACHE & VISUAL DEFECTS | NA | PITUITARY MACROADENOMA | EXCISE PITUTARY MASS | PITUITARY MACROADENOMA | MULTIPLE 1X0.8X0.6 CM |
| 206 | 862813 | 14719/14720 -19 | SS | 30 | M | FEVER & SEIZURES | NA | RIGHT CEREBELLAR ABSCESS | RIGHT FRONTOPARIETAL | HYPERINTENCE LESION WITH EDEMA | MULTIPLE 2X1X1 CM |
| 207 | 863776 | 14987-19 | NS | 72 | M | HEADACHE,SEIZURES & VISUAL DEFECTS | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | MULTIPLE 3X2X1.5 CM |
| 208 | 863838 | 14948-19 | RK | 63 | F | HEADACHE ,SEIZURES & LEFT SIDED WEAKNESS | NA | ? HIGH GRADE GLIOMA | RIGHT FRONTO PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 6X5X4 CM |
| 209 | 868614 | 15130-19 | BS | 60 | M | HEADACHE & SEIZURES | K/C/O HTN | INTRACRANIAL HEMORRHAGE | RIGHT FRONTAL | NA | MULTIPLE 5X3X2 CM |
| 210 | 868539 | 15184-19 | MG | 64 | M | HEADACHE & SEIZURES | NA | HIGH GRADE GLIOMA | LEFT FRONTAL | HIGH GRADE GLIOMA - GLIOBLASTOMA | MULTIPLE 7X5X2 CM |
| 211 | 825757 | 15557-19 | JS | 64 | M | HEADACHE & RIGHT SIDED WEAKNESS | NA | ? GLIOBLASTOMA | LEFT PARIETAL | HIGH GRADE GLIOMA - GLIOBLASTOMA | MULTIPLE 2.5X2X0.5 CM |
| 212 | 876531 | 228-20 | HK | 62 | F | HEADACHE & SEIZURES | NA | LEFT FRONTAL SOL | LEFT FRONTAL | SOL IN LEFT FRONTAL | SINGLE 7X6X2 CM |
| 213 | 855953 | 484-20 | JK | 55 | F | LEFT SIDED WEAKNESS | K/C/O HTN HYPERTHYROIDISM | RIGHT PARIETAL GLIOBLASTOMA | RIGHT PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 4X3X2 CM |
| 214 | 865620 | 1196-20 | SK | 34 | M | HEADACHE & SEIZURES | NA | LOW GRADE GLIOMA | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 5.5X5X1 CM |
| 215 | 893262 | 1376-20 | HS | 21 | M | LEFT SIDE WEAKNESS & HEADACHE | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 7X6X2 CM |
| 216 | 893089 | 1490-20 | NS | 30 | M | HEADACHE & VISION DEFECT | NA | PITUITARY ADENOMA | EXCISE PITUTARY MASS | PITUTARY MACROADENOMA | MULTIPLE 1X0.8X0.3 CM |
| 217 | 875063 | 1644-20 | BS | 65 | M | LEFT SIDED WEAKNESS | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 3X2X1 CM |
| 218 | 898146 | 1882-20 | NK | 65 | F | HEADACHE & SEIZURES | NA | BRAIN SOL | RIGHT TEMPORAL | HYPERDENSE MASS WITH EDEMA | MULTIPLE 14X10X4.5 CM |
| 219 | 905106 | 2114-20 | JS | 65 | M | RIGHT SIDED WEAKNESS & VERTIGO | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 2X1X1 CM |
| 220 | 906902 | 2416-20 | SK | 55 | F | HEADACHE & SEIZURES | NA | MENINGIOMA | LEFT FRONTOPARIETAL | SOL IN LEFT FRONTOPARIETAL | MULTIPLE 7X4X2 CM |
| 221 | 907073 | 2501-20 | MC | 64 | F | HEADACHE & VISUAL DISTURBANCE | NA | SPHENOID WING MENINGIOMA | SPHENO ORBITAL FISSURE | MASS LESIONS IN MIDDLE CRANIAL FOSSA | MULTIPLE 5X3X1 CM |
| 222 | 909284 | 2538-20 | SS | 78 | M | HEADACHE & VERTIGO | NA | OLFACTORY GROOVE SOL | OLFACTORY GROOVE LEISON | ISOTANE LEISON IN THE OLFACTORY GROOVE | MULTIPLE 8X5X1 CM |
| 223 | 912831 | 2716-20 | SR | 60 | F | DIFFICULTY IN WALKING AND HEADACHE | NA | CEREBELLAR SOL | RIGHT CEREBELLUM | ISOINTENSE LESION IN THE CEREBELLAR | MULTIPLE 9X5.8X2.4 CM |
| 224 | 911747 | 2746-20 | K | 50 | F | HEADACHE , VERTIGO & RIGHT SIDED WEAKNESS | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 5X4X3 CM |
| 225 | 914081 | 2792-20 | J | 17 | F | HEADACHE & SEIZURES | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | RIGHT TEMPORAL EDEMA | MULTIPLE 1.5X 1X0.8 CM |
| 226 | 899218 | 2913-20 | VS | 65 | F | HEADACHE & SEIZURES | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 6X4X3 CM |
| 227 | 918047 | 3229-20 | BN | 65 | M | LEFT SIDED WEAKNESS & HEADACHE | NA | RIGHT PARIETAL GLIOBLASTOMA | RIGHT PARIETAL LESION | POSSIBLY HIGH GRADE GLIOMA - GLIOBLASTOMA | MULTIPLE 4X3X2 CM |
| 228 | 908689 | 3304-20 | S | 25 | M | LEFT SIDED WEAKNESS & HEADACHE | NA | RIGHT TEMPOROPARIETAL | RIGHT TEMPORAL | HYPERDENSE AREA IN THE RIGHT TEMPOROPARIETAL | MULTIPLE 4X3X2 CM |
| 229 | 922267 | 3807-20 | SK | 54 | F | HEADACHE & FEVER | NA | LEFT TEMPORAL ABSCESS | LEFT TEMPORAL | LEFT TEMPORAL ABSCESS | MULTIPLE 1.1X0.7X0.5 CM |
| 230 | 926377 | 3830-20 | SS | 65 | M | RIGHT SIDED WEAKNESS & HEADACHE | NA | LEFT PARIETAL SOL | LEFT PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 4X2X1 CM |
| 231 | 857290 | 3885-20 | RV | 65 | M | LEFT SIDED WEAKNESS | NA | RIGHT BRAIN SOL | RIGHT FRONTOPARIETAL | HIGH GRADE GLIOMA | MULTIPLE 5X4X2.5 CM |
| 232 | 928491 | 4046-20 | SS | 65 | M | HEADACHE , VERTIGO & SEIZURES | NA | LEFT TEMPORAL PARIETAL SOL | LEFT TEMPOROPARIETAL | HIGH GRADE GLIOMA | MULTIPLE 3.5X3X0.8 CM |
| 233 | 928095 | 4070-20 | MA | 58 | M | SEIZURES | K/C/O CA LUNG | LEFT PARIETAL SOL | LEFT PARIETAL | HYPERDENSE AREA IN LEFT PARIETAL | TWO 4X3.5X3 CM |
| 234 | 927608 | 4128-20 | CD | 42 | F | HEADACHE & SEIZURES | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 11X7.5X4 CM |
| 235 | 451879 | 4336-20 | PK | 55 | F | HEADACHE | K/C/O CA BREAST | CEREBELLAR SOL | CEREBELLAR | CEREBELLAR SOL | MULTIPLE 4X3X2 CM |
| 236 | 937683 | 4818-20 | SS | 55 | M | HEADACHE & SEIZURES | K/C/O HYPERTENSION | RIGHT TEMPORAL ICH | RIGHT TEMPORAL | NA | MULTIPLE 1.5X1X0.5 CM |
| 237 | 431992 | 5508-20 | KR | 66 | F | HEADACHE & SEIZURES | ULCERATIVE COLITIS | RIGHT FRONTAL SOL | RIGHT FRONTAL | HYPOINTENSE ENHANCING LESION IN RIGHT FRONTAL | MULTIPLE 1X0.5X0.3 CM |
| 238 | 700508 | 5533-20 | GK | 66 | F | LEFT SIDED WEAKNESS & HEADACHE | NA | RIGHT PARIETO OCCIPITAL MENINGIOMA | RIGHT PARIETO OCCIPITAL | MENINGIOMA | MULTIPLE 8X5X2 CM |
| 239 | 946093 | 5612-20 | AB | 52 | M | HEADACHE | NA | HIGH GRADE GLIOMA | LEFT PARIETO OCCIPITAL | LEFT PARIETO OCCIPITAL SOL | MULTIPLE 5X3X1.5 CM |
| 240 | 941155 | 5746-20 | APK | 55 | F | FEVER , HEADACHE & SEIZURES | SINUSITIS | SUBDURAL EMPYEMA | RIGHT PARIETAL | DURAL THICKENING | MULTIPLE 9X7X2 CM |
| 241 | 951774 | 5943-20 | S | 80 | M | WEAKNESS OF RIGHT LOWER LIMBS | NA | LEFT PARIETAL SOL | LEFT PARIETAL | HIGH GRADE GLIOMA | MULTIPLE 3X2.8X0.7 CM |
| 242 | 936353 | 5922-20 | KK | 65 | F | HEADACHE & RIGHT SIDED WEAKNESS | NA | LEFT FRONTAL SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 4X3X1.5 CM |
| 243 | 959497 | 6255-20 | JC | 44 | F | HEADACHE & VISION DEFECT | NA | PITUITARY MACROADENOMA | EXCISE PITUTARY MASS | PITUITARY ADENOMA | SINGLE 0.8X0.5X0.1 CM |
| 244 | 960652 | 6298-20 | K | 31 | F | DIFFICULTY IN WALKING & SEIZURES | NA | LEFT PARIETAL SOL | LEFT PARIETAL | LEFT PARIETAL | MULTIPLE 4X2X1.5 CM |
| 245 | 5640 | 6849-20 | PK | 66 | M | HEADACHE & SEIZURES | NA | LEFT FRONTAL SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 12X8X4 CM |
| 246 | 972427 | 6879-20 | RP | 80 | M | FEVER & HEADACHE | NA | BRAIN SOL | DURAL BIOPSY | NA | MULTIPLE 4X2.8X1 CM |
| 247 | 974688 | 7027-20 | GV | 68 | M | SEIZURES | NA | FRONTAL SOL | LEFT FRONTAL | HIGH GRADE GLIOMA | MULTIPLE 7X5X2 CM |
| 248 | 979576 | 7374-20 | TS | 70 | M | SEIZURES & HEADACHE | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 6.5X4.5X2.5 CM |
| 249 | 982926 | 7624-20 | M | 33 | M | HEADACHE & SEIZURES | RECURRENT ASTROCYTOMA | FRONTAL SOL | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 6X5X2.5 CM |
| 250 | 989250 | 8036-20 | BS | 70 | M | HEADACHE & SEIZURES | NA | RIGHT FRONTOTEMPORAL SOL | RIGHT FRONTOTEMPORAL | HIGH GRADE GLIOMA | MULTIPLE 6X3X2 CM |
| 251 | 959615 | 8389-20 | HS | 52 | M | SEIZURES | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | RIGHT FRONTAL SOL | MULTIPLE 7X6X2 CM |
| 252 | 1000050 | 8685-20 | US | 60 | M | HEADACHE & DIFFICULTY IN WALKING | K/C/O HTN | RIGHT PARIETAL HEMATOMA | RIGHT PARIETAL | RIGHT PARIETAL HEMATOMA | MULTIPLE 6X4X1.5 CM |
| 253 | 995177 | 8782-20 | AKG | 54 | M | UNEXPLAINED NEUROLOGICAL DEFICIT | NA | RIGHT TEMPORAL HEMATOMA | RIGHT TEMPORAL | RIGHT TEMPORAL HEMATOMA | MULTIPLE 5X4X2 CM |
| 254 | 995519 | 78-21 | GK | 32 | F | DIFFICULTY IN WALKING AND SEIZUERS | NA | RIGHT TEMPORAL SOL | RIGHT TEMPORAL | ICH RIGHT TEMPORAL | MULTIPLE PIECES 2X1.5X1 CM |
| 255 | 1009099 | 185-21 | RK | 42 | F | HEADACHE | NA | LEFT PARIETAL SOL | LEFT PARIETAL | LEFT PARIETAL SOL ?GLIOMA | MULTIPLE PIECES 6X4X2 CM |
| 256 | 1008971 | 236-21 | PL | 57 | M | SEIZURES RIGHT SIDE WEAKNESS | NA | HIGH GRADE GLIOMA | LEFT TEMPORAL | HIGH GRADE GLIOMA-?GLIOBLASTOMA | MULTIPLE 5X3X1 CM |
| 257 | 1011174 | 326-21 | GS | 26 | M | HEADACHE | NA | MENINGIOMA | RIGHT TEMPORAL | RIGHT TEMPORAL SOL ? MENINGIOMA | MULTIPLE 7X5X3 CM |
| 258 | 1118966 | 601-21 | PJ | 14 | M | HEADCHE & SEIZURES | NA | LEFT CEREBELLAR SOL | LEFT CEREBELLUM | LEFT CEREBELLAR SOL | MULTIPLE 2X1.5X.0.6 CM |
| 259 | 1026264 | 1469-21 | BS | 50 | M | HEADACHE & SEIZURES | NA | LEFT TEMPORAL SOL | LEFT TEMPORAL | LEFT TEMPORAL SOL ?LOW GRADE GLIOMA | MULTIPLE 4X2X1 CM |
| 260 | 1026915 | 1558-21 | GS | 48 | M | HEADACHE | NA | RETRO OCCIPITAL MENINGIOMA | RETRO OCCEPT | RETRO OCCIPITALSOL ? MENINGIOMA | MULTIPLE 8X4X2 CM |
| 261 | 1033573 | 1769-21 | NS | 84 | M | HEADACHE & SEIZURES | NA | ? GLIOBLASTOMA LEFT PARIETAL | LEFT FRONTOPARIETAL | HIGH GRADE GLIOMA-?GLIOBLASTOMA | MULTIPLE 8.5X6X3 CM |
| 262 | 1055036 | 2652-21 | RK | 58 | M | LEFT HEMIPLEGIA | NA | RIGHT PARIETAL SOL | RIGHT PARIETAL | ?GLIOMA | MULTIPLE 5X3X2 CM |
| 263 | 1079480 | 3280-21/3348-21 | VK | 53 | M | HEADACHE | NA | ? HIGH GRADE GLIOMA | RIGHT PARIETAL | HIGH GRADE GLIOMA ? GLIOBLASTOMA | MULTIPLE 3X2X1 CM |
| 264 | 1088051 | 3608-21 | LK | 52 | F | HEADACHE & SEIZURES | NA | LEFT TEMPORAL FRONTAL SOL | LEFT TEMPOROFRONTAL | LEFT TEMPOROFRONTAL SOL ? MENINGIOMA | MULTIPLE 5X3X2 CM |
| 265 | 1112202 | 4114-21 | MK | 55 | M | DROWSINESS AND HEADACHE | NA | RIGHT FRONTAL SOL | RIGHT FRONTAL | GLIOBLASTOMA IN RIGHT FRONTAL | MULTIPLE 4X3X1.5 CM |
| 266 | 1114286 | 4353-21 | SS | 30 | M | SEIZURES RIGHT FRONTAL HEADACHE | NA | LOW GRADE GLIOMA | RIGHT FRONTAL | LOW GRADE GLIOMA | MULTIPLE 5.5X4X2 CM |

| SR. NO. | MRD | MICROSCOPIC EXAMINATION | SPECIAL STAINS | | | | |
|---|---|---|---|---|---|---|---|
| | | | AFB | PAS | GMS | RETICULIN | PERL'S |
| 186 | 773387 | HISTOMORPHOLOGICAL FEATURES ARE CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | RETICULIN LOSS | NA |
| 187 | 771609 | TENSIVE AREA OF NECROSIS. TUMOR CELLS ARE ARRANGED IN DIFFUSE SHEETS AND PALISADED AROUND AREAS OF NECROSIS. FOCAL AREAS SHOW FIBRILLARY BACKGROUND WITH PRESENCE OF TUMOR GIANT CELLS. IN SOME AREAS THERE IS PRESENCE OF TUMOR CELLS HAVING ROUND NUCLEI, ABUNDANT AMOUNT OF CYTOPLASM WITH PERINUCLEAR HALO | NA | NA | NA | NA | NA |
| 188 | 782242 | PROMINENT CYSTIC CHANGE IDENTIFIED ALONG WITH AREA OF HEMORRHAGE AND FOCI OF NECROSIS. MANY OF THE BLOOD VESSELS HAVE HYALINIZED WALLS. EXTENSIVE AREAS OF CALCIFICATION NOTED. OCCASIONAL MITOTIC FIGURE SEEN. | NA | NA | NA | NA | NA |
| 189 | 775656 | W PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS AND CORDS. THESE TUMOR CELLS ARE ROUND TO OVAL HAVE HYPERCHROMATIC NUCLEI, INCONSPICUOS NUCLEOLI AND SCANT AMOUNT OF CYTOPLASM. THESE CELLULAR AREAS ARE SURROUNDED BY THE NODULAR PALE ISLANDS WHICH HAVE REDUCED CELLULARITY AND A FIBRIL | NA | NA | NA | RETICULIN FREE PALE ISLANDS | NA |
| 190 | 782229 | TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS ARE SMALL TO SPINDLE CELL IN SHAPE, PLEOMORPHIC VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. ENDOTHELIAL CELL PROLIFERATION SEEN. THE TUMOR CELLS ARE SEEN PALISADING AROUND CENTRAL AREA OF NECROSIS. M | NA | NA | NA | NA | NA |
| 191 | 785937 | F FASCICLES AND FOCALLY IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI IN FEW WITH MODERATE CYTOPLASM AND ILL DEFINED CYTOPLASMIC BOUNDARIES. NUMEROUS VASCULAR CHANNELS ARE NOTED WITH CONGESTION AND HAEMORRHAGE. FEW OF THE VESSELS ALSO SHOW HYALINISATION OF THE WALL. MITOTIC | NA | NA | NA | NA | NA |
| 192 | 788160 | OW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. TUMOR CELLS HAVE PLEOMORPHIC NUCLEI WITH MODERATE AMOUNT OF CYTOPLASM. MARKED NUCLEAR ATYPIA WITH ATYPICAL MITOSIS SEEN. AREAS OF HEMORRHAGE AND NECROSIS ALSO SEEN. ENDOTHELIAL CELL PROLIFERATION IS ALS | NA | NA | NA | NA | NA |
| 193 | 789534 | AKEN FROM BOTH THE BIOPSIES SHOW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED AGAINST A FIBRILLARY BACKGROUND. TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI WITH ILL DEFINED CYTOPLASMIC BOUNDARIES. THERE IS PRESENCE OF MITOTIC FIGURES WITH PROMINENT ENDOTHELIAL CELL PROLIFERATION AND OCCASIONAL FOCUS OF NE | NA | NA | NA | NA | NA |
| 194 | 792537 | CEREBELLAR SHOW A WELL ENCAPSULATED TUMOR HAVING TWO COMPONENT. STROMAL COMPONENT IS SEEN AS LARGE POLYGONAL CELLS WITH VACUOLATED CLEAR CYTOPLASM AND VASCULAR COMPONENT IS SEEN AS VARIABLY SIZED THIN WALLED CHANNELS LINED BY ENDOTHELIAL CELLS. | NA | NA | NA | NA | NA |
| 195 | 796026 | ULAR TUMOR WITH TUMOR CELL PRESENT IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE CYTOPLASM. ENDOTHELIAL CELL PROLIFERATION ALONG WITH EXTENSIVE AREAS OF NECROSIS SEEN. TUMOR GIANT CELLS NOTED. THE TUMOR IS INFILTRATING | NA | NA | NA | NA | NA |
| 196 | 796346 | ANGED IN CORDS AND WHORLS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. FEW OF THESE CELLS HAVE EOSINOPHILIC CYTOPLASM WITH PRESENCE OF MUCIN IN THE BACKGROUND. AN OCCASIONAL MITOTIC FIGURE SEEN ALONG WITH FOCI OF NECROSIS. NO SMALL CELL CHANGE/ SHEETIN | NA | NA | NA | NA | NA |
| 197 | 801209 | E OF CELLULAR TUMOR WITH CELLS ARRANGED IN WHORLS, SHEETS AND FASCICLES. THE TUMOR CELLS HAVE OVAL TO ELONGATED VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CYTOPLASMIC BOUNDARIES.NO SIGNIFICANT MITOSIS/NECROSIS/SMALL CELL CHANGE SEEN IN THE SECTIONS EXAMINED.NO BRAIN PARENCHYMA INCL | NA | NA | NA | NA | NA |
| 198 | 405782 | SECTIONS SHOW PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN NESTS, SHEETS AND FOCAL PAPILLARY ARCHITECTURE. THE TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEI AND SCANT TO MODERATE AMOUNT OF CYTOPLASM. EXTENSIVE AREAS OF HEMORRHAGE AND NECROSIS SEEN. | NA | NA | NA | NA | NA |
| 199 | 811690 | SECTION TAKEN FROM DEEP BRAIN PARENCHYMA SHOWS EXTENSIVE AREAS OF NECROSIS WITH INFILTRATION BY TUMOR CELLS ARRANGED IN NESTS AND SMALL CLUSTERS. INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. | NA | NA | NA | NA | NA |
| 200 | 813699 | HISTOMORPHOLOGICAL FEATURES ARE CONSISTENT WITH PITUITARY ADENOMA. | NA | NA | NA | RETICULIN LOSS | NA |
| 201 | 816047 | SECTIONS SHOW PRESENCE OF DURA MATTER HAVING NUMEROUS COLLECTIONS OF EPITHELIOID CELLS FORMING GRANULOMA WITH FOREIGN BODY AND LANGHANS GIANT CELL REACTION SURROUNDED BY LYMPHOCYTIC COLLAR.NO CASEOUS NECROSIS/FUNGAL HYPHAE OR SPORES/EVIDENCE OF MALIGNANCY SEEN. | NEGATIVE | NEGATIVE | NEGATIVE | NA | NA |
| 202 | 827318 | CTIONS SHOW AN INFILTRATIVE CELLULAR TUMOR WITH PREDOMINANTLY MONOMORPHIC ROUND CELLS WITH ROUND NUCLEI AND CLEAR CYTOPLASM. DELICATE BRANCHING CAPILLARIES IN CHICKEN WIRE PATTERN ALSO SEEN IN THE TUMOR AREA. PERINEURONAL SATELLITOSIS SEEN.NO SIGNIFICANT ATYPIA/MITOSIS/NECROSIS/ENDOTHELIAL PROLIFERATION NOT | NA | NA | NA | NA | NA |
| 203 | 815469 | SECTION TAKEN FROM BRAIN SHOWS PRESENCE OF TUMOR ARRANGED IN SHEETS. MANY OF THE CELLS HAVE OVAL TO ELONGATED NUCLEI WITH PROMINENT NUCLEOLI. MANY VASCULAR CHANNELS NOTED IN THE BACKGROUND. FEW OF THE CELLS EXHIBIT FOAMY CYTOPLASM. THESE CELLS ARE EXHIBITING NUCLEAR ATYPIA. | NA | NA | NA | NA | NA |
| 204 | 838215 | SECTIONS SHOW PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. OCCASIONAL GIANT CELLS ALSO SEEN AND AREAS OF HAEMORRHAGE IDENTIFIED. | NA | NA | NA | NA | NA |
| 205 | 841193 | HISTOLOGY CONSISTENT WITH PITUITARY ADENOMA | NA | NA | NA | RETICULIN LOSS | NA |
| 206 | 862813 | SECTION TAKEN FROM BOTH THE BIOPSIES SHOW AREAS OF NECROSIS CONTAINING SHEETS OF FOAMY MACROPHAGES ADMIXED WITH NUMEROUS NEUTROPHILS. THE SURROUNDING BRAIN PARENCHYMA SHOWS NUMEROUS COLLECTIONS OF NEUTROPHILS FORMING MIACROABSCESS. | NEGATIVE | NA | NEGATIVE | NA | NA |
| 207 | 863776 | SECTION SHOW PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND NUCLEI, VESICULAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. | NA | NA | NA | RETICULIN LOSS | NA |
| 208 | 863838 | RANGED IN SHEETS. THE TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM.MANY BIZARRE FORM AND TUMOR GIANT CELLS SEEN ALONG WITH ATYPICAL MITOTIC FIGURES SEEN. FEW GEMISTIOCYTES ALSO SEEN. ALSO SEEN IS ENDOTHELIAL CELL PROLIFERATION FORMING GLOMERULOID STRUCTURES AND FOCI OF NECRO | NA | NA | NA | NA | NA |
| 209 | 868614 | SECTIONS TAKEN FROM THE TISSUE SENT AS TISSUE WITH BLOOD VESSELS SHOW VARIABLY THICKENED CONGESTED AND DILATED VASCULAR CHANNELS. FEW OF THE VESSELS SHOW FOCI OF DYSTROPHIC CALCIFICATION. | NA | NA | NA | NA | NA |
| 210 | 868539 | OW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. TUMOR CELLS HAVE PLEOMORPHIC HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. MANY GEMISTIOCYTES AND BIZARRE FORMS SEEN WITH FEW TUMOR GIANT CELLS. FOCI OF NECROSIS AND ENDOTHELIAL CELL PROLIFERATIO | NA | NA | NA | NA | NA |
| 211 | 825757 | EOMORPHIC TUMOR WITH CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. FEW GEMISTOCYTES AND NUMEROUS TUMOR GIANT CELLS SEEN. NUMEROUS MITOSIS FIGURES SEEN.ENDOTHELIAL CELL PROLIFERATION ALSO IDENTIFIED. NORMAL BRA | NA | NA | NA | NA | NA |
| 212 | 876531 | F A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS SHOW ROUND HYPERCHROMATIC NUCLEI AND CLEAR AMOUNT OF CYTOPLASM WITH DISTINCT CELL BOUNDARIES. CHICKEN WIRE VASCULATURE SEEN. THE TUMOR IS INFILTRATING INTO ADJACENT BRAIN PARENCHYMA. NO SIGNIFICANT NUCLEAR ATYPIA/MITOSIS/NECROSIS/ END | NA | NA | NA | NA | NA |
| 213 | 855953 | SENCE OF SHEETS OF CELLS AGAINST FIBRILLARY BACKGROUND. THESE CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF EOSINOPHILIC CYTOPLASM AND ARE PALISADE AROUND AREA OF NECROSIS. IN OTHER AREA THERE IS PRESENCE OF A SPINDLE CELLS HAVING HYPERCHROMATIC NUCLEI AND ILL DEFINED CYTOPLASMIC BOUNDARIES. TU | NA | NA | NA | HIGHLIGHTS SPINDLE CELL AREA | NA |
| 214 | 865620 | EH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI WITH PERINUCLEAR CLEARING AT PLACES. PROMINENT CYSTIC CHANGE AND CHICKEN WIRE VASCULATIVE NOTED AT PLACES. THE TUMOR IS INFILTRATING THE ADJACENT OF BRAIN PARENCHYMA AND REACHING UPTO SUBPIAL LOCATION.NO MITOSIS/NEC | NA | NA | NA | NA | NA |
| 215 | 893262 | LLS ARRANGED IN SHEETS INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI WITH PERINUCLEAR CLEARING AT PLACES. PROMINENT CYSTIC CHANGE AND CHICKEN WIRE VASCULATURE NOTED. ADJACENT NORMAL BRAIN PARENCHYMA IS INFILTRATED BY TUMOR CELLS WITH PROMINENT PERINEURAL SATELLITOSIS.NO SIGNIFICANT MITOSIS/ NECROSIS/ EI | NA | NA | NA | NA | NA |
| 216 | 893089 | SECTIONS SHOW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND NUCLEI, VESICULAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. LARGE AREAS OF HAEMORRHAGIC ALSO NOTED. | NA | NA | NA | RETICULIN LOSS | NA |
| 217 | 875063 | LS ARRANGED IN SHEETS.TUMOR CELLS EXHIBIT MARKED NUCLEAR ATYPIA, HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM AGAINST A FIBRILLARY BACKGROUND. AREAS OF NECROSIS AND ENDOTHELIAL CELL PROLIFERATION NOTED. BRISK MITOSIS SEEN. FEW MULTINUCLEATED TUMOR GIANT CELLS AND GEMISTOCYTES ARE ALSO EVIDENT. | NA | NA | NA | NA | NA |
| 218 | 898146 | S AND EXTRADURAL TUMOR SHOWS PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN NODULES. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI, INCONSPICOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. THESE NODULE ARE SURROUNDED BY LYMPHOCYTIC INFILTRATE. MITOTIC FIGURES NOTED. TUMOR IS INFILTR | NA | NA | NA | NA | NA |
| 219 | 905106 | CTIONS SHOW PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI, INCONSPICUOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. BRISK MITOSIS WITH AREAS OF NECROSIS AND ENDOTHELIAL CELL PROLIFERATION ALSO NOT | NA | NA | NA | NA | NA |
| 220 | 906902 | OR CELLS ARE ARRANGED IN WHORLS AND NESTS. INDIVIDUAL TUMOR CELLS ARE OVAL IN SHAPE WITH DELICATE CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CELL BOUNDARIES, NUCLEAR GROOVES AND PSAMMOMA BODIES SEEN, HOWEVER, NO NUCLEAR ATYPIA/BRISK MITOSIS/NECROSIS/SMALL CELL CHANGE/SHEETING PATTERN ID | NA | NA | NA | NA | NA |
| 221 | 907073 | TUMOR CELLS ARRANGED IN WHORLS AND NESTS. INDIVIDUAL TUMOR CELLS ARE OVAL IN SHAPE AND HAVE DELICATE NUCLEAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CYTOPLASM BOUNDARIES. AREA OF COLLAGENISATION AND PSAMMOMA BODIES NOTED.NO NUCLEAR ATYPIA/BRISK MITOSIS/NECROSIS/SMALL CELL CHANGE/SH | NA | NA | NA | NA | NA |
| 222 | 909284 | UMOR WITH TUMOR CELLS ARRANGED IN SYNCYTIAL PATTERN AND FEW WHORLS. INDIVIDUAL TUMOR CELLS ARE OVAL IN SHAPE HAVING DELICATE CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM WITH ILL-DEFINED CELL BOUNDARIES. SMALL AMOUNT OF BRAIN PARENCHYMA ALSO IDENTIFIED SHOWING NEST OF TUMOR CELLS -INVASION. FOCI OF NECROSIS | NA | NA | NA | NA | NA |
| 223 | 912831 | ES. INDIVIDUAL TUMOR CELLS HAVE SPINDLE SHAPED NUCLEI, VESICULAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. A SMALL FOCI OF NECROSIS AND HEMORRHAGE ALSO NOTED. MITOSIS UPTO 4/10HPF SEEN.NO BRAIN PARENCHYMA SEEN IN THE SECTIONS EXAMINED. SECTIONS TAKEN FROM THE BONY TISSUE SHOWS NO TUMOR INFILTRATION.NO N | NA | NA | NA | NA | NA |
| 224 | 911747 | IN SHEETS AND FASCICLES AGAINST FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI WITH PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. FOCALLY MANY TUMOR CELLS HAVE SPINDLE SHAPED NUCLEI WITH ILL DEFINED CYTOPLASMIC BOUNDARIES- ? SARCOMA .NUMEROUS GEMISTOCYTES, BRISK MITOSIS AREA A | NA | NA | NA | NA | NA |
| 225 | 914081 | SECTION SHOW NORMAL LOOKING BRAIN PARENCHYMA ALONG WITH AREAS OF HEMORRHAGE.ALSO SEEN ARE MANY THICK WALLED DILATED AND CONGESTED BLOOD VESSELS HAVING VARIABLE WALL THICKNESS.NO EPITHELIOID CELL GRANULOMA/NECROSIS/EVIDENCE OF MALIGNANCY SEEN. | NA | NA | NA | NA | NA |
| 226 | 899218 | NCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN DIFFUSE SHEETS.TUMOR CELLS EXHIBIT MARKED NUCLEAR PLEOMORPHISM, HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. ALSO IDENTIFIED ARE NUMEROUS GEMISTIOCYTES.BRISK MITOSIS NOTED WITH PRESENCE OF ATYPICAL FORMS. AREAS OF NECROSIS SEEN.ENDOTHELIAL CELL | NA | NA | NA | NA | NA |
| 227 | 918047 | UMOR WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. AREAS OF NECROSIS SURROUNDED BY TUMOR CELLS , ENDOTHELIAL CELL PROLIFERATION AND AREAS OF HEMORRHAGE ALSO NOTED. BRISK MITOSIS SEEN. TUMOR CELLS ARE ALSO INFILTRATING THE NORMAL | NA | NA | NA | NA | NA |
| 228 | 908689 | SECTIONS SHOW BRAIN PARENCHYMA HAVING AN AREA OF INTRAPARENCHYMAL HEMORRHAGE. ALSO SEEN NUMEROUS THICK WALL BLOOD VESSELS. MANY OF THEM HAVE VARIABLY IRREGULAR THICKENED WALLS. OCCASIONAL VESSEL SHOW THROMBI WITH WALL SHOWING ACUTE INFLAMMATORY INFILTRATE. NO FIBRINOID NECROSIS SEEN. | NA | NA | NA | NA | NA |
| 229 | 922267 | LARGE AREAS OF NECROSIS HAVING SHEETS OF INFLAMMATORY CELLS MAINLY NEUTROPHILS ADMIXED WITH FEW FOAMY MACROPHAGES.NO FUNGAL HYPHAE OR SPORES/GRANULOMA/EVIDENCE OF MALIGNANCY SEEN IN MULTIPLE SECTIONS EXAMINED.SECTION TAKEN FROM THE TISSUE SENT AS TEMPORAL BONE SHOW TRABECULAE HAVING FOCAL LOSS OF C | NEGATIVE | NEGATIVE | NEGATIVE | NA | NA |
| 230 | 926377 | ETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE MODERATE NUCLEAR PLEOMORPHISM, HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. BRISK MITOTIC ACTIVITY NOTED. MICROVASCULAR PROLIFERATION AND EXTENSIVE AREAS OF HEMORRHAGE AND NECROSIS SEEN. ALSO IDENTIFIED ARE FEW BITS OF NORMAL B | NA | NA | NA | NA | NA |
| 231 | 857290 | WITH CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND TO OVAL HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. PROMINENT CYSTIC CHANGE, BRISK MITOSIS, MICROVASCULAR PROLIFERATION AND EXTENSIVE AREAS OF NECROSIS NOTED. BRAIN PARENCHYMA INCLUDED IN THE SHOWS INFILTRATION BY THE TUMOR C | NA | NA | NA | NA | NA |
| 232 | 928491 | W PRESENCE OF A CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS. TUMOR CELLS EXHIBIT MODERATE NUCLEAR PLEOMORPHISM, HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. NUMEROUS MITOSIS, MICROVESICULAR PROLIFERATION AND AREAS OF HEMORRHAGE AND NECROSIS SEEN. NORMAL BRAIN PARENCHYMA INFILTRA | NA | NA | NA | NA | NA |
| 233 | 928095 | D IN PAPILLAE, NESTS AND GLANDS. INDIVIDUAL TUMOR CELL HAVE MODERATE NUCLEAR PLEOMORPHISM, VESICULAR CHROMATIN, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. MULTINUCLEATE AND BIZARRE FORMS ALSO SEEN. ALSO SEEN FEW AREAS WITH NECROSIS AND HEMORRHAGE. NO FIBRILLARY BACKGROUND NOTED. TUMOR IS INFI | NA | NA | NA | NA | NA |
| 234 | 927608 | A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBITS MODERATE NUCLEAR PLEOMORPHISM AND HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. MANY GEMISTIOCYTES (> 20%) WITH FEW GIANT CELLS SEEN. OCCASIONAL MITOTIC FIGURE ALSO NOTED. ADJACENT BRAIN PARENCHYMA IS ALSO INFILTRATED BY | NA | NA | NA | NA | NA |
| 235 | 451879 | TIONS FROM CELEBELLUM .SHOWS PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN NESTS AND SHEETS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. MITOTIC FIGURES NOTED. AREAS OF NECROSIS ALSO SEEN. THE TUMOR IS INFILTRATING INTO THE ADJACENT BRAIN PARENCH | NA | NA | NA | NA | NA |
| 236 | 937683 | SECTIONS FROM BRAIN TISSUE SHOW PRESENCE OF INTRAPARENCHYMAL HEMORRHAGE. NO EVIDENCE OF MALIGNANCY SEEN IN SECTIONS EXAMINED | NA | NA | NA | NA | NA |
| 237 | 431992 | TIONS FROM BRAIN PARENCHYMA SHOW PRESENCE OF A CELLULAR TUMOR WITH CELLS ARRANGED IN SHEETS AND AROUND THE VESSELS. INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI, PROMINENT NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. NUMEROUS APOPTOTIC BODIES AND OCCASIONAL MITOSIS SEEN. ADJACENT BRAIN SHOWS CYSTIC CHA | NA | NA | NA | NA | NA |
| 238 | 700508 | SHEETS. INDIVIDUAL TUMOR CELLS ARE OVAL IN SHAPE HAVING DELICATE CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM WITH ILL DEFINED CYTOPLASM BOUNDAIRES MANY COLLECTIONS OF FOAMY MACROPHAGES AND CHOLESTEROL CLEFT IDENTIFIED. OCCASIONAL MITOSIS NOTED (3/10HPF) ? FOCUS OF INVASION SEEN INTO ADJACENT BRAIN PARENCHY | NA | NA | NA | NA | NA |
| 239 | 946093 | R CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND HYPERCHROMATIC NUCLEI WITH PERINEURAL CLEARING AND MODERATE CYTOPLASM. CHICKEN WIRE VASCULATURE, PERINEURONAL SATELLETOSIS AND CYSTIC CHANGE A LSO SEEN. PROMINENT MICROVASCULAR PROLIFERATION, BRISK MITOSIS (>6/10 HPF) & EXTENSIVE AREAS OF NECROS | NA | NA | NA | NA | NA |
| 240 | 941155 | FLAMMATORY GRANULATION ACUTE AND CHRONIC. ALSO SEEN ARE BONY TRABECULAE HAVING FOCAL LOSS OF OSTEOCYTES.SECTIONS FROM SENT AS SUBDURAL MEMBRANE SHOW FIBROCOLLAGENOUS TISSUE HAVING FLORID INFLAMMATORY GRANULATION ACUTE AND CHRONIC NON SPECIFIC. AREAS OF NECROSIS SEEN.NO WELL FORMED EPITHELIOID CELL G | NEGATIVE | NA | NEGATIVE | NA | NA |
| 241 | 951774 | TUMOR CELLS ARRANGED IN DIFFUSE SHEETS AND FOCALLY IN PERIVASCULAR ARRANGEMENT. INDIVIDUAL TUMOR CELLS HAVE ROUND VESICULAR NUCLEI, INCONSPICOUS NUCLEOLI AND MODERATE AMOUNT OF CYTOPLASM. PROMINENT CYSTIC CHANGE, NECROSIS WITH PALLISADED TUMOR CELLS AND NUMEROUS TUMOR GIANT CELLS ALSO NOTED. BRISK MIT | NA | NA | NA | NA | NA |
| 242 | 936353 | F A CELLULAR TUMOR CELLS WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS EXHIBITS MARKED PLEOMORPHISM, VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. PROMINENT CYSTIC CHANGE WITH EXTENSIVE MICROVASCULAR PROLIFERATION NOTED. ALSO SEEN ARE AREAS OF NECROSIS SURROUNDED BY TUMOR CELLS. BRIS | NA | NA | NA | NA | NA |
| 243 | 959497 | SECTION SHOW PRESENCE OF CELLULAR TUMOR WITH TUMOR CELLS ARRANGED IN SHEETS. INDIVIDUAL TUMOR CELLS HAVE ROUND NUCLEI, VESICULAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. | NA | NA | NA | RETICULIN LOSS | NA |
| 244 | 960652 | R CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE ROUND MONOMORPHIC HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. ALSO NOTED PERIVASCULAR PSEUDOROSETTES, GEOGRAPHICAL AREAS OF NECROSIS AND HEMORRHAGE. OCCASIONAL MITOSIS SEEN. FOCAL CLEAR CELL CHANGE | NA | NA | NA | NA | NA |
| 245 | 5640 | MOR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE MILD NUCLEAR PLEOMORPHISM, VESICULAR CHROMATIN AND MODERATE AMOUNT OF CYTOPLASM. BRISK MITOTIC ACTIVITY, CYSTIC CHANGE, PALLISADING NECROSIS AND NUMEROUS GEMISTIOCYTIC ALSO SEEN. TUMOR CELLS ARE ALSO INFILTRATING INTO T | NA | NA | NA | NA | NA |
| 246 | 972427 | AS DURAL MEMBRANE SHOWS DURAMATER WITH COLLECTIONS OF INFLAMMATORY CELLS SEEN AS NEUTROPHILS AND FEW LYMPHOCYTES. AREAS OF NECROSIS AND SHEETS OF HEMOSIDERIN LADEN MACROPHAGES ALSO NOTED.HOWEVER, NO WELL FORMED EPITHELIOID CELL GRANULOMA/FUNGAL HYPHAE OR SPORES EVIDENCE OF MALIGNANCY SEEN IN THI | NEGATIVE | NEGATIVE | NA | NA | NA |
| 247 | 974688 | ELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE MODERATE NUCLEAR PLEOMORPHISM, VESICULAR CHROMATIN AND MODERATE CYTOPLASM. EXTENSIVE AREAS OF NECROSIS, PROMINENT MICROVASCULAR PROLIFERATION, TUMOR GIANT CELLS AND BRISK MITOTIC ACTIVITY NOTED. TUMOR CELLS ARE ALSO INFIL | NA | NA | NA | NA | NA |
| 248 | 979576 | OR CELLS ARRANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE PLEOMORPHIC VESICULAR NUCLEI, PROMINENT NUCLEOLI, MODERATE AMOUNT OF CYTOPLASM. ATYPICAL MITOTIC ACTIVITY NOTED. GEOGRAPHICAL AND PALISADED NECROSIS, PROMINENT MICROVASCULAR PROLIFERATION AND CYSTIC CHANGE ALSO SEEN. T | NA | NA | NA | NA | NA |
| 249 | 982926 | RANGED IN SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS HAVE MILD NUCLEAR PLEOMORPHISM, VESICULAR NUCLEI AND MODERATE AMOUNT OF CYTOPLASM.PROMINENT CYSTIC CHANGE AND OCCASIONAL MITOTIC FIGURES NOTED.TUMOR CELLS ARE ALSO SEEN INFILTRATING INTO THE ADJACENT NORMAL BRAIN PARENCHYMA.HOWEV | NA | NA | NA | NA | NA |
| 250 | 989250 | TUMOR CELLS ARRANGED IN DIFFUSE SHEETS AGAINST A FIBRILLARY BACKGROUND. INDIVIDUAL TUMOR CELLS EXHIBIT MODERATE PLEOMORPHISM HAVING HYPERCHROMATIC NUCLEI AND MODERATE AMOUNT OF CYTOPLASM. BRISK MITOSIS AND ENDOTHELIAL CELL PROLIFERATION SEEN. FOCI OF NECROSIS PALISADED BY TUMOR CELLS ALSO IDENTIFIED. TUMOR | NA | NA | NA | NA | NA |
| 251 | 959615 | OR CELLS ARE ROUND HAVING VESICULAR NUCLEI, PERINUCLEAR HALO AND MODERATE AMOUNT OF CYTOPLASM. INTERSPERSED ARE SEEN NUMEROUS DELICATE BRANCHING CAPILLARIES. PROMINENT CYSTIC CHANGE AND SHEETS OF MINIGEMISTIOCYTS SEEN AT PLACES. MITOTIC FIGURES NOTED (<6/10 HPF).TUMOR CELLS ARE ALSO INFILTRATING THE ADJACE | NA | NA | NA | NA | NA |
| 252 | 1000050 | CONTAINER 2 SHOW BRAIN PARENCHYMA EXHIBITING LARGE INTRAPARENCHYMAL HEMORRHAGE WITH FOCI OF CYSTIC CHANGE. ALSO SEEN ARE NUMEROUS THICK WALLED BLOOD VESSELS WITH AN OCCASIONAL ONES SHOWING IRREGULAR THICKENING OF VESSEL WALL. EXTENSIVE AREAS OF HEMORRHAGE SEEN.HOWEVER, NO EVIDENCE OF MALIGNANCY SE | NA | NA | NA | NA | NA |
| 253 | 995177 | HOCYTES AND PLASMA CELLS. THE ADJACENT BRAIN PARENCHYMA SHOWS EXTENSIVE AREAS OF NECROSIS WHICH ARE INFILTRATED BY SHEETS OF FOAMY MACROPHAGES ADMIXED WITH LYMPHOCYTES AND PLASMA CELLS. MANY OF THE BLOOD VESSELS ARE THICKENED AND EXPANDED WITH DENSE INFILTRATION OF WALL BY NEUTROPHILS, LYMPHOCYTES AN | NEGATIVE | NEGATIVE | NA | NA | NA |
| 254 | 995519 | SHOWS BRAIN PARENCHYMA WITH FOCI OF INTRAPARENCHYMAL HEMORRHAGE. AREAS OF NECROSIS INFILTRATED BY SHEETS OF GITTER CELLS ALSO IDENTIFIED.FEW OF THE HISTIOCYTES SHOW FEATURES OF ERYTHROPHAGOCYTOSIS. NO EPITHELOID CELL GRANULOMA/FUNGAL HYPHAE OR SPORES/ EVIDENCE OF MALIGNANCY IDENTIFIED IN MULTIPLE SECTION | NA | NEGATIVE | NA | NA | NA |
| 255 | 1009099 | EAR PLEOMORPHISM, HYPERCHROMATIC NUCLEI, ELONGATED CYTOPLASMIC PROCESSES AND MODERATE CYTOPLASM. PROMINENT CYSTIC CHANGE AND TINY FOCUS OF NECROSIS SEEN.FOCALLY ARE PRESENT SHEETS OF GEMISTOCYTES AND MANY BLOOD VESSELS WITH HYALINISED WALLS. TUMOR CELLS ARE ALSO SEEN INFILTRATING THE ADJACENT NORMA | NA | NA | NA | NA | NA |
| 256 | 1008971 | WITH TUMOR CELLS ARRANGED IN SHEETS AND FASCICLE AGAINST A FIBRILLARY BACKGROUND.INDIVIDUAL TUMOR CELLS HAVE VESICULAR NUCLEI AND M

| SR. NO. | MRD | CONGO RED | IMMUNO-HISTOCHEMISTRY | | | | | | | | | FINAL HISTOPATHOLOGICAL DIAGNOSIS | GRADE | IDH STATUS | CATEGORY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | GFAP | ATRX | IDH-1 | MIB-1 | P53 | CK | EMA | S100 | OTHER | | | | |
| 186 | 773387 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 187 | 771609 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 188 | 782242 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GANGLIOGLIOMA | I | NA | NEOPLASTIC |
| 189 | 775656 | NA | NEGATIVE | NA | NA | NA | NA | NA | NEGATIVE | POSITIVE | CD56,SYNAPTOPHYSIN,NSE,CD99 - POSITIVE | DESMOPLASTIC MEDULLOBLASTOMA | IV | NA | NEOPLASTIC |
| 190 | 782229 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 191 | 785937 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 192 | 788160 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 193 | 789534 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 194 | 792537 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | HAEMANGIOBLASTOMA | I | NA | NEOPLASTIC |
| 195 | 796026 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 196 | 796346 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | CHORDOID MENINGIOMA | II | NA | NEOPLASTIC |
| 197 | 801209 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 198 | 405782 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | METASTATIC CARCINOMATOUS DEPOSITS | NA | NA | NEOPLASTIC |
| 199 | 811690 | NA | NA | NA | NA | NA | NA | NA | NA | NA | CK,CK7,CK-20, P63, CEA, UROPLAKIN-II - POSITIVE & TTF-1,GATA3,SATB2,P40 - NEGATIVE | METASTATIC CARCINOMATOUS DEPOSITS LIKELY FROM URINARY TRACT | NA | NA | NEOPLASTIC |
| 200 | 813699 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 201 | 816047 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GRANULOMATOUS INFLAMMATION - POSSIBLY TB | NA | NA | NON-NEOPLASTIC |
| 202 | 827318 | NA | NEGATIVE | POSITIVE | POSITIVE | 8% | NEGATIVE | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | MUTANT | NEOPLASTIC |
| 203 | 815469 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 204 | 838215 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 205 | 841193 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 206 | 862813 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ABSCESS - NON SPECIFIC | NA | NA | NON-NEOPLASTIC |
| 207 | 863776 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 208 | 863838 | NA | POSITIVE | POSITIVE | NEGATIVE | 40% | POSITIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 209 | 868614 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 210 | 868539 | NA | POSITIVE | NEGATIVE | POSITIVE | 30% | NEGATIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | MUTANT | NEOPLASTIC |
| 211 | 825757 | NA | POSITIVE | POSITIVE | NEGATIVE | 40% | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 212 | 876531 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 213 | 855953 | NA | POSITIVE | POSITIVE | NEGATIVE | 30% | POSITIVE | NA | NA | NA | VIMENTIN POSITIVE & EMA NEGATIVE | GLIOSARCOMA | IV | WILD TYPE | NEOPLASTIC |
| 214 | 865620 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 215 | 893262 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 216 | 893089 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 217 | 875063 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 218 | 898146 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | NON HODGKIN LYMPHOMA - DLBCL | NA | NA | NEOPLASTIC |
| 219 | 905106 | NA | POSITIVE | POSITIVE | NEGATIVE | 50% | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 220 | 906902 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 221 | 907073 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 222 | 909284 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ATYPICAL MENINGIOMA | II | NA | NEOPLASTIC |
| 223 | 912831 | NA | NA | NA | NA | 25% | NA | POSITIVE | POSITIVE | POSITIVE | NA | ATYPICAL MENINGIOMA | II | NA | NEOPLASTIC |
| 224 | 911747 | NA | POSITIVE | POSITIVE | NEGATIVE | 30% | NA | NA | NA | NA | VIMENTIN POSITIVE | GLIOSARCOMA | IV | WILD TYPE | NEOPLASTIC |
| 225 | 914081 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 226 | 899218 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 227 | 918047 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 228 | 908689 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 229 | 922267 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ABSCESS NON SPECIFIC | NA | NA | NON-NEOPLASTIC |
| 230 | 926377 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 231 | 857290 | NA | POSITIVE | POSITIVE | NEGATIVE | 25% | POSITIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 232 | 928491 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 233 | 928095 | NA | NA | NA | NA | NA | NA | NA | NA | NA | CK7, TTF-1 POSITIVE CK20,CDX-2 NEGATIVE | METASTATIC ADENOCARCINOMATOUS DEPOSITS - LIKELY LUNG | NA | NA | NEOPLASTIC |
| 234 | 927608 | NA | POSITIVE | POSITIVE | NEGATIVE | 4% | POSITIVE | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | WILD TYPE | NEOPLASTIC |
| 235 | 451879 | NA | NA | NA | NA | NA | NA | NA | NA | NA | ER,PR,HER2 neu POSITIVE | METASTATIC CARCINOMATOUS DEPOSITS - PRIMARY BREAST | NA | NA | NEOPLASTIC |
| 236 | 937683 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | INTRAPARENCHYMAL HEMORRHAGE | NA | NA | NON-NEOPLASTIC |
| 237 | 431992 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | NON HODGKIN LYMPHOMA - DLBCL | NA | NA | NEOPLASTIC |
| 238 | 700508 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | MENINGOTHELIAL MENINGIOMA | I | NA | NEOPLASTIC |
| 239 | 946093 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ANAPLASTIC OLIGODENDROGLIOMA | III | NA | NEOPLASTIC |
| 240 | 941155 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PACHYMENINGITIS | NA | NA | NON-NEOPLASTIC |
| 241 | 951774 | NA | POSITIVE | NEGATIVE | NEGATIVE | 70% | POSITIVE | NA | NA | NA | NA | GIANT CELL GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 242 | 936353 | NA | POSITIVE | POSITIVE | NEGATIVE | 30% | NEGATIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 243 | 959497 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PITUITARY ADENOMA | NA | NA | NEOPLASTIC |
| 244 | 960652 | NA | POSITIVE | POSITIVE | NEGATIVE | 20% | POSITIVE | NA | NA | NA | NA | ANAPLASTIC EPENDYMOMA | III | NA | NEOPLASTIC |
| 245 | 5640 | NA | POSITIVE | POSITIVE | NEGATIVE | 35% | POSITIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 246 | 972427 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | PACHYMENINGITIS | NA | NA | NON-NEOPLASTIC |
| 247 | 974688 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 248 | 979576 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | GLIOBLASTOMA | IV | NA | NEOPLASTIC |
| 249 | 982926 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | NA | NEOPLASTIC |
| 250 | 989250 | NA | POSITIVE | POSITIVE | NEGATIVE | 40% | POSITIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 251 | 959615 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 252 | 1000050 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ARTERIO-VENOUS MALFORMATION | NA | NA | NON-NEOPLASTIC |
| 253 | 995177 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | BRAIN INFARCT WITH FEATURES OF MENINGITIS & VASCULITIS | NA | NA | NON-NEOPLASTIC |
| 254 | 995519 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | INFARCT BRAIN | NA | NA | NON-NEOPLASTIC |
| 255 | 1009099 | NA | POSITIVE | NEGATIVE | POSITIVE | 10% | POSITIVE | NA | NA | NA | NA | ANAPLASTIC ASTROCYTOMA | III | MUTANT | NEOPLASTIC |
| 256 | 1008971 | NA | POSITIVE | POSITIVE | NEGATIVE | 30% | POSITIVE | NA | NA | NA | VIMENTIN POSITIVE | GLIOSARCOMA | IV | WILD TYPE | NEOPLASTIC |
| 257 | 1011174 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | ATYPICAL MENINGIOMA | II | NA | NEOPLASTIC |
| 258 | 1118966 | NA | POSITIVE | POSITIVE | NEGATIVE | 2% | NA | NA | NA | NA | NA | PILOCYTIC ASTROCYTOMA | I | NA | NEOPLASTIC |
| 259 | 1026264 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | OLIGODENDROGLIOMA | II | NA | NEOPLASTIC |
| 260 | 1026915 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 261 | 1033573 | NA | POSITIVE | POSITIVE | NEGAIVE | 20% | POSITIVE | NA | NA | NA | NA | GIANT CELL GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 262 | 1055036 | NA | NA | NA | NA | NA | NA | NA | NA | NA | LCA,CD4, CD3, CD8, CD20,CD30 POSITIVE AND CD15 IS NEGATIVE | LYMPHOMATOID GRANULOMATOSIS | I | NA | NEOPLASTIC |
| 263 | 1079480 | NA | POSITIVE | POSITIVE | NEGATIVE | 35% | POSITIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 264 | 1088051 | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | TRANSITIONAL MENINGIOMA | I | NA | NEOPLASTIC |
| 265 | 1112202 | NA | POSITIVE | POSITIVE | NEGATIVE | 15% | POSITIVE | NA | NA | NA | NA | GLIOBLASTOMA | IV | WILD TYPE | NEOPLASTIC |
| 266 | 1114286 | NA | POSITIVE | NEGATIVE | POSITIVE | 3% | NA | NA | NA | NA | NA | DIFFUSE ASTROCYTOMA | II | MUTANT | NEOPLASTIC |

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Date 20/4/2021

The Principal,
DMC,Ludhiana.

**Subject:** **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Dr.Gagandeep Singh**, a student of your college for **MD Pathology** along with the names of Supervisor/Co-Supervisor as under:-

**Topic Plan of Thesis**
Histopathological study of intracranial lesions

Supervisor
Dr. Harpreet Kaur
Prof.,
Dept. of Pathology,
DMCH, Ludhiana.

Co-Supervisor
Dr. Kavita Saggar
Prof. & Head,
Dept. of Radio-diagnosis,
DMCH, Ludhiana.

Dr. Vikram Narang
Assoc. Prof.,
Dept. of Pathology,

The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,( if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

*Note :*

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

# HISTOPATHOLOGICAL STUDY OF INTRACRANIAL LESIONS

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

PATHOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

NOVEMBER, 2019

DR. GAGANDEEP SINGH

DEPARTMENT OF PATHOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

## ABSTRACT OF PLAN OF THESIS

| | |
|---|---|
| Title | HISTOPATHOLOGICAL STUDY OF INTRACRANIAL LESIONS |
| For the degree of | MD PATHOLOGY |
| Name of the candidate | Dr. Gagandeep Singh |
| Supervisor | Dr. Harpreet Kaur, Professor, Department of Pathology, DMCH, Ludhiana |
| Co-supervisor(s) | Dr. Kavita Saggar, Professor & Head, Department of Radiodiagnosis, DMCH, Ludhiana<br>Dr. Vikram Narang, Associate Professor, Department of Pathology, DMCH, Ludhiana |
| Institution | Dayanand Medical College and Hospital, Ludhiana |
| University | Baba Farid University of Health Sciences, Faridkot |

**Introduction**: This study will be conducted for a period of 5 years (3.5 years retrospective & 1.5 years prospective) in the Department of Pathology, Dayanand Medical College and Hospital. All the intracranial biopsies received in pathology department will be included in the study for histomorphological spectrum.

**Aims & Objectives**: To study various histological patterns of intracranial lesions and to correlate histopathological diagnosis with radiological findings.

**Material & Methods**: This study will be conducted in the Department of Pathology at Dayanand Medical College and Hospital, Ludhiana for a period of 5 years (3.5 years retrospective & 1.5 years prospective) w.e.f. 1st July 2016 to 31th December 2019 (Retrospective) and from 1st January 2020 to 30th June 2021 (Prospective). All the intracranial biopsies received in pathology department will be processed and stained with Hematoxylin and Eosin (H&E). Sections will be analysed under the light microscope. Special stains and IHC markers will be done if needed. Histopathological diagnosis will be correlated with radiological findings.

**Clinical Significance**: To obtain a reasonable good database for a more accurate assessment and understanding of the histological patterns of central nervous system lesions.

**Keywords**: Intracranial lesions, Brain tumours.

| (Signature) | (Signature) | (Signature) | (Signature) | (Signature) |
|---|---|---|---|---|
| Candidate | Co-supervisor | Co-supervisor | Supervisor | Principal |

## CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"HISTOPATHOLOGICAL STUDY OF INTRACRANIAL LESIONS"** exist in the Department of Pathology, DMCH, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself under our supervision and guidance.

**Supervisor**

**Dr. Harpreet Kaur**
Professor
Department of Pathology
DMCH, Ludhiana

**Co-Supervisor(s)**

**Dr. Kavita Saggar**
Professor & Head
Department of Radiodiagnosis
DMCH, Ludhiana

**Dr. Vikram Narang**
Associate Professor
Department of Pathology
DMCH, Ludhiana

**Head of the Department**

**Dr. Neena Sood**
Professor & Head
Department of Pathology
DMCH, Ludhiana

Place : LUDHIANA

Date : 9/12/19

## APPROVAL PROFORMA
## BY
## RESEARCH & ETHICAL COMMITTEE
## DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

| | |
|---|---|
| **Name of candidate** | Dr. Gagandeep Singh |
| **Department** | Pathology |
| **Topic of Thesis** | HISTOPATHOLOGICAL STUDY OF INTRACRANIAL LESIONS |
| **Likely date of appearing in PG examination** | May 2022 |
| **Date of enrollment** | May, 2019 |
| **Name of Head of Department** | Dr. Neena Sood |
| • **Supervisor**<br>• **Co-Supervisor(s)** | Dr. Harpreet kaur<br>Dr. Kavita Saggar<br>Dr. Vikram Narang |
| **Signature of Members of Research Committee with Stamp** | **Signature of Members of Ethics Committee with Stamp** |
| Dr. Jaspal Singh<br>(Professor & Head, Surgery) | Mr. A K Jindal<br>(Member, Ethics Committee) |
| Dr. Dinesh Gupta<br>(Professor & Head, Medicine) | Dr. Anu Sharma<br>(Professor, Anatomy) |
| Dr. Sandeep Kaushal<br>(Assistant Dean Academics) | Dr. Deepinder K Chhina<br>(Professor & Head, Microbiology) |
| Dr. Rajoo Singh Chhina<br>(Dean Academics) | Dr. Neena Sood<br>(Professor & Head, Pathology) |
| Dr. Gurpreet S Wander<br>(Vice Principal) | Dr. Gurpreet S Wander<br>(Vice Principal) |
| | Dr. Gagandeep Singh<br>(Secretary, Ethics Committee<br>Professor & Head, Neurology) |

**Approved: Yes / ~~No~~**

**Dr. Sandeep Puri**
**Chairperson**
**Research Committee**

Chairperson
Research Committee

**Approved: Yes / ~~No~~**

**Prof. Arvind Malhotra**
**Chairperson**
**Ethics Committee**

Chairperson
Ethics Committee

# BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

## PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

| | | |
|---|---|---|
| 1 | NAME OF THE CANDIDATE AND ADDRESS | Dr. Gagandeep Singh<br>4249, Durga Puri, Haibowal Kalan,<br>Ludhiana<br>Punjab<br>Pin-141001 |
| 2 | NAME OF THE INSTITUTE | Dayanand Medical College and Hospital, Ludhiana |
| 3 | COURSE OF STUDY AND SUBJECT | MD Pathology |
| 4 | DATE OF ADMISSION OF COURSE | 13-05-2019 |
| 5 | TITLE OF THE TOPIC | HISTOPATHOLOGICAL STUDY OF INTRACRANIAL LESIONS |

## 6. BRIEF RESUME OF THE INTENDED WORK

### 6.1 NEED FOR THE STUDY

Intracranial space occupying lesions (ICSOL) range in etiology from neoplastic, infective, inflammatory, vascular to trauma. The space occupying lesions in central nervous system (CNS) cause grave life threatening outcome irrespective of their nature. The reason behind is that these lesions grow in a confined space and are present close to vital structures.[1] Therefore, it is of great importance to establish the accurate diagnosis for timely neurosurgical intervention.

The space occupying lesions (SOLs) of CNS are most commonly due to tumors that represent 1-2% tumours in adults. Effect of such brain lesions may be local, indicating location of lesion. Slow growing SOLs can be accomodated by atrophy of adjacent brain tissue. Rapid enlargement causes rise in pressure of affected compartment. Hence the CNS lesions require immediate neurosurgical intervention.[2]

Early diagnosis is necessary to plan appropriate intervention. Radiological investigative modalities like computed tomography [CT] and Magnetic Resonance Imaging [MRI] are important preoperative diagnostic tools to detect the lesion.[3] Therefore advances in neuroimaging during the last 20 years have revolutionized the diagnosis and treatment of central nervous system diseases. The diagnosis and treatment of patients with CNS lesions has improved because of these advances and has allowed access to regions that were previously inaccessible. These new approaches have placed the pathologist in a key role in the diagnosis and treatment of patients with central nervous system lesions.

Although brain biopsy is an invasive procedure, every effort should be made to establish an accurate diagnosis pre-operatively. However, many patients with intracranial lesions may have atypical presentation secondary to intratumoral haemorrhage, arterial occlusion, cerebral infarction or tumor involvement of silent areas. Such lesions pose a diagnostic challenge and need pre-operative radiological evaluation.[3]

Therefore interpretation of histopathology of CNS lesions is significantly aided when viewed in perspective of clinical, per-operative and radiological findings. Radiological findings can be used by pathologists to narrow down the differential diagnoses. However, radiological diagnosis of SOL requires confirmation by histopathology. Hence, histopathology is considered as gold standard for diagnosis of CNS lesions.

The present study aims to provide preliminary data on morphological patterns of intracranial space occupying lesions in North Indian region and to study histopathological spectrum with correlation of radiological findings (where ever possible) of ICSOL.'s.

## 6.2 REVIEW OF LITERATURE

The central nervous system composed of brain and spinal cord, is invested with meninges and suspended in fluid, the cerebrospinal fluid (CSF) which is produced by specialized choroid plexus structures. Macroscopically, all parts of the CNS are made up of grey matter and white matter. Grey matter contains most of the neurone cell bodies and their dendritic processes while the white matter contains the axons.

The term "intracranial space occupying lesions" is defined as any lesion, benign or malignant, primary or secondary, as well as any inflammatory or parasitic mass lying within the cranial cavity. It also includes haematomas, different types of cysts, & vascular malformations.[4] Intracranial space occupying lesions are important cause of neurological morbidity. Their potentially life threatening behavior results from their growing in a confined space as well as their proximity to vital structures.

Early diagnosis is necessary to plan appropriate intervention. Radiological investigative modalities like computed tomography [CT] and Magnetic resonance Imaging [MRI] are important preoperative diagnostic tools to detect the lesion.[3] The goals of diagnostic imaging in the patient with suspected intracranial masses include detection of the presence of a mass, localization of the extent of the mass (including definition of involvement of key structures & assessment of the presence & severity of secondary changes, e.g edema, herniation, hemorrhage)-characterization of the nature of the process.

For the evaluation of CNS lesions, correlation of clinical, radiologic, and histologic data and knowledge of clinicopathologic entities and their common locations are required.

Some studies have been undertaken to prove the utility of histological profiling with radilogic correlation in diagnosing CNS lesions.

Patil and Karandikar did a study of 124 cases of intracranial and intraspinal space occupying lesions in 2019. Intraoperative diagnosis was made in 70 cases. The final diagnoses in all the cases were made by routine histopathology according to classification and grading of CNS tumors WHO 2016. Neoplastic lesions comprised 83.88% (104

cases), including metastatic tumors, while 16.12% (20 cases) were nonneoplastic. Astrocytic tumors constituted the largest category. Incidence was more in females than males and in the fifth and sixth decades of life. This study highlighted histological diversity of CNS tumors in adults and children.[5,6]

Sunila et al in 2018 analysed 88 cases of ICSOL's. Histopathological evaluation was done from biopsy samples sent in formalin and stained by H&E. Histopathological diagnosis of cases included neoplastic lesions [90.9%] and non-neoplastic lesions [9.09%].[3] Correct diagnosis was achieved by radiology in 36 cases [45%] by complete concordance. For the detection of neoplastic lesions, radiological diagnosis had sensitivity of 96.25%, specificity of 50% and efficacy of 92.04%. The study showed radiological investigation to be a reliable diagnostic tool for space occupying central nervous system lesions.[7]

Similarly Adnan H.A. et al in 2017 studied a total of 185 CNS lesions. The diagnosis was made on routinely processed tissue and stained by H&E. Out of 185 CNS lesions analysed, 107 (58%) were males and 78 (42%) were females. minimum age of case was 8 years and maximum was 75 years. The most common lesions were of neoplastic in nature among which glioma was more common (35%), followed by meningioma (23%).[8] Other lesions included fungal infections, pituitary adenoma, schwannoma, neurofibroma, carotid body paraganglioma, craniopharyngioma, arachnoid cyst, medulloblastoma and medulloepithelioma. This study gives histological spectrum of non-neoplastic and neoplastic CNS lesions.[9]

B.N. Kumarguru et al conducted a study in 2017 to analyse various histopathological patterns of CNS lesions and to evaluate the association with the distribution of ABO blood groups with respect to a control group. Out of 147 cases included in the study, neoplastic lesions (84.35%) constituted the majority, which included neuroepithelial tumours (29.25%) as predominant pattern. Non-neoplastic lesions constituted only (15.64%), which included inflammatory lesion (8.16%) as the predominant pattern. ABO blood group data was available in 92 cases (84.4%) of neoplastic lesions,

which included 71 cases (48.29%) of primary CNS neoplasms. Blood group O was the most frequent blood group in neoplastic lesions (40.21%). The association between the CNS neoplasms and ABO blood groups was not statistically significant, but a definite change in the pattern of distribution of ABO blood groups was observed between neoplastic lesions and control groups.[10,11]

Similarly a study done by Renu Thambi et al in 2017 a total of 510 cases of brain tumours were studied over a period of seven years and the analysis showed that most of the brain tumours occurred between 40-60 years of age, with a male to female ratio of 0.9:1. [2] Majority of cases involved dura and cerebral lobes except for the occipital lobe, and meningioma and glial tumours were the most common broad histological types. WHO grade IV tumours and metastasis were common in males as compared to females.[12]

In a prospective study by Neha Jindal et al done in 2016, 80 patients with ICSOLs were studied by CT & MRI. Imaging findings were evaluated with the clinical findings & histopathological findings (wherever available). Most patients were in age range of 31-50 years (mean 42.2 y). Male:females ratio was 3:2. Solitary lesions were present in 58 patients (72.5%) and multiple lesions in 22 patients (27.5%). 76.2% lesions were supratentorial & 23.7% were infratentorial in location. 76.2% patients were having neoplastic lesions and 23.7% had non-neoplastic lesions. Metastases were the most common neoplastic lesion while among non neoplastic lesions, arachnoid cysts were the most common. For neoplastic lesions the imaging sensitivity was 90%, specificity was 57%, accuracy was 84.2%, positive predictive value (PPV) was 90% and neagative predicted value (NPV) was 57%. While for the non neoplastic lesions imaging sensitivity was 57%, specificity was 90%. accuracy was 84.2%, PPV was 57% and NPV was 90% (95% CI). The study concluded that these modalities helped in the early diagnosis & localization of the SOL and in complement with advanced neurosurgical techniques, brightened the prognosis of mass lesions.[4,13]

In 2016, Jat KC et al studied 59 patients with CNS tumors to analyze their histopathological profiles. Patients ages ranged from 3.5-65 years with a mean age of 41.2 years and a male to female ratio of 1.8:1. They found out that frontal lobe was the predominant (37.3%) affected region. Most tumors originated from neuroepithelium (67.8%) followed by meninges (22.03%). The most frequent type of CNS tumour in this study was astrocytoma, followed by meningioma. Males were at much higher risk of developing CNS tumour in comparison to females (1.8:1). Therefore the exact histological diagnosis of CNS tumors was essential to predict the prognostic factors.[14,15]

Dogar T. et al in 2015 included 102 consecutive cases of ICSOLs. Their clinical history and radiological findings were noted. The histopathological diagnosis was correlated with radiological diagnosis, and study showed that histopathological diagnosis remains more definitive as compared to radiological diagnosis in the context of ICSOLs of brain.[1,16]

Nibhoria, et al in 2015 conducted a 5 years study in a single tertiary care center for patients clinically diagnosed with CNS tumors. Study shows that overall tumors of neuroepithelial tissue (51.7%) were the most common entity followed by the tumors of meninges (34.8%), metastatic tumors (5.6%), tumors of peripheral nerves (4.5%), tumors of the sellar region (2.3%), and lymphomas and hematopoietic neoplasm (1.1%). Also Rising global trends in the incidence of CNS tumors, irrespective of age have been observed. The study highlighted the histological diversity in CNS tumors in both, adults as well as pediatric age groups.[17]

## 6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To study various histological patterns of Intracranial lesions.
2. To correlate histopathological diagnosis with radiological findings (wherever possible).

## 7. MATERIALS AND METHODS

### 7.1 Source of Data

Biopsies of intracranial lesions received in the Department of Pathology of Dayanand Medical College and Hospital, Ludhiana over a period of 5 years, that is one and half years prospective (1st January 2020 to 30th June 2021) and three and a half years retrospectively (1st July 2016 to 31st December 2019) will be analysed.

### 7.2 METHOD OF COLLECTION OF DATA

**INCLUSION CRITERIA**

- All specimens from cranial vault.

**EXCLUSION CRITERIA-**

- Biopsies of cranial nerves, peripheral nerves and spinal cord.

**PROCEDURE**

Tissue biopsies from intracranial lesions will be processed and Hematoxylin and Eosin (H&E) staining will done. The sections will be analysed under the light microscope.

**STAINING PROCEDURE**

1. Stain rehydrated sections in Hematoxylin solution for 20-40 minutes.
2. Wash in tap water for 1-5 minutes, until sections turn blue ("bluing").
3. Differentiate sections in 70% ethanol—containing 1% HCl—for 5 seconds. This removes excess dye, allowing nuclear details to emerge.
4. Wash 1-5 minutes in tap water until blue.
5. Stain in Eosin solution for 10 minutes.
6. Wash 1-5 minutes in tap water,
7. Dehydrate, clear and mount.
8. Special Stains (AFB, PAS, GMS, Reticulin etc) and immuno histochemistry will be done wherever required.

**DURATION OF STUDY**

This study will be conducted for a period of 5 years (3.5. years retrospective & 1.5 years prospective) in the Department of Pathology, Dayanand Medical College and Hospital. All the intacranial biopsies received in the Pathology Department will be included in the study for histomorphological spectrum.

**OUTCOME**

To obtain a reasonable good database for a more accurate assessment and understanding of the histological patterns of intracranial lesions.

**STATISTICAL ANALYSIS**

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

**7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.**

YES, Radiological investigations

**7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?**

Attached herewith

## 8.0 REFERENCES


1. Dogar T, Imran AA, Hasan M, Jaffar R, Bajwa R, Qureshi ID. Space occupying lesions of central nervous system: a radiological and histopathological correlation. Biomedica. 2015;31:15-20.
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10. Kumarguru BN, Pallavi P, Sunila, Manjunath GV, Vasan TS, Rajalakshmi BR. Histopathological study of central nervous system lesions: Emphasizing association of neoplasms with ABO blood groups. Journal of Clinical and Diagnostic Research. 2017;11:EC15-20.
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14. Jat KC, Vyas SP, Bihari NA, Mehra K. Central nervous system tumors: a histopathological study. Int J Res Med Sci. 2016;4:1539-45.
15. Louis DN, Ohgaki H, Wiestler OD, Cavenee WK, Burger PC, Jouvet A et al. WHO Classification of Tumors of the Central Nervous System. 2007;114:97-109.
16. Jaffer R, Dogar T, Qureshy A, Qureshi N. Central ner-vous system tumours – A study of frequency and mor-phology. JFJMC, 2011;05:116-8.
17. Nibhoria S, Tiwana KK, Phutela R, Bajaj A, chhabra S, Bansal S. Histopathological spectrum of central nervous system tumors: A Single centre study of 100 cases. International Journal of Scientific Study. 2015;3:130-4.

## PROFORMA

Serial No._..... Histopath No. ..... MRD no....

Name..... Age..... Sex.....

Date ..... Phone no..... Address.....

Chief complaints:

Past History:

Family History:

Clinical Diagnosis:

Investigations-

1. Hematological: Hemoglobin- TLC-

DLC- ESR-

2. Coagulation Profile: PT- INR-

PTTK- Ratio-

3. Liver function tests: Bilirubin (T)- Bilirubin (D)-

SGOT- SGPT-

ALP- T. Proteins-

Albumin-

**Signature of Candidate** **Signature of Supervisor**

4. Renal function tests: Urea- Creatinine-

Sodium(Na) Potassium(k) Chloride(cl)

X-ray/CT/MRI findings (if available)

Site of Biopsy:-

Gross examination of the specimen received:-

Microscopic examination:-

Special stains (AFB, PAS, GMS, Reticulin etc)

Immuno-histochemistry (if done):

Final Histopathological Diagnosis:

**Signature of Candidate** **Signature of Supervisor**